Caritas Bianchi College of Careers
Access Provided by:
SILVERCHAIR

Chapter 5: Lymphedema

Marisa Perdomo; Rose L. Hamm

INTRODUCTION

CHAPTER OBJECTIVES

At the end of this chapter, the learner will be able to:

1. **Differentiate between the components of the lymphatic and the venous system.**
2. **Relate the function of each lymph system component to the formation of lymphedema.**
3. **Diagnose lymphedema according to cause, pathophysiology, and stage.**
4. **Differentiate between lymphedema and chronic venous insufficiency.**
5. **Define the components of Starling's Law and describe their role in lymphatic flow.**
6. **Select the appropriate compression therapy for a patient with peripheral lymphedema.**
7. **Design an exercise program for a patient with lymphedema.**
8. **Discuss the principles of manual lymphatic mobilization, as well as the indications and contraindications.**
9. **Educate patients on skin care and strategies to prevent recurrent lymphedema complications.**

Lymphedema is a chronic inflammatory condition that develops as a result of lymphatic insufficiency. Lymphatic insufficiency occurs from a decrease in reabsorption or a decrease in transport capacity of the lymphatic system. It can be primary malformation of the lymph system or an acquired condition due to obstruction or damage to the system (**TABLE 5-1**).

TABLE 5-1

Primary and Secondary Lymphedema: Causes, Onset, and Characteristics

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Caritas Bianchi College of Careers
Access Provided by:
SILVERCHAIR INFORMATION/SYSTEMS

Primary Lymphedema Causes	Onset	Secondary Lymphedema Causes	Onset
Inherited genetic mutations resulting in abnormal development of lymph vessels: • Hypoplasia • Hyperplasia • Aplasia • Kinmonth syndrome (inguinal lymph node fibrosis) Genetic mutations with autosomal dominant pattern: • Chromosome 5q34-q35 • FLT4 • FOXC2 • VEGFR3 • SOX18	Variable age onset depending upon the gene involved*	Damage to lymph nodes and/or lymph vessels that results in decreased lymphatic reabsorption and transportation (mechanical insufficiency) Trauma Radiation therapy Tumor obstruction Infection CVI	Any time after damage to lymph nodes and vessels: Either soon after lymph node dissection *or* months, years, *or* decades later Depends on patient medical history, co-morbidities (number of lymph nodes removed, radiation therapy, chemotherapy, venous pathologies, and obesity), and general health
Milroy's disease (congenital lymphedema) FOXC2 VEGEFR3 SOX18*	Onset during infancy*	Surgery Trauma Radiation therapy Tumor obstruction (cancer) Infection CVI	Depends on patient medical history, comorbidities, and general health
Meige's disease (lymphedema praecox) MIM153200*	Onset in childhood or around puberty; may begin in early 20s or 30s		
Lymphedema tarda	Onset at 35 years or later		

*Primary and secondary lymphedema differ in causes and onset, which are used to make a differential diagnosis; however, the characteristics are very similar—an edematous limb, skin changes, loss of joint spaces. There are some characteristics of hereditary lymphedema, however, that distinguish it from secondary lymphedema. These include but are not limited to hypoparathyroidism, microcephaly, intestinal lymphangiectasia, ptosis, yellow nails, pleural effusions, cerebral arteriovenous anomalies, distichiasis, congenital heart defects, and webbing of the neck.

Data from Connell F, Brice G, Jeffery S, Keeley V, Mortimer P, Mansour S. A new classification system for primary lymphatic dysplasias based on phenotype.*Clin Genet.* 2010;77:438–452; Levinson KL, et al. Age of onset in hereditary lymphedema. *J Pediatr.* 2003;142:704–708; Rizzo C. Lymphedema praecox. *Derm Online J.* 2009;15(8):7.

The lymphatic system is interrelated with all of the other systems of the body. Its primary roles include conducting immune system surveillance, assisting

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

the cardiovascular system to maintain fluid homeostasis, and aiding the digestive system in the breakdown of long-chain fatty acids. The immunological functions involve both the immediate response to pathogens and the long-term resistance to repeated exposure to pathogens.

ANATOMY OF THE LYMPH SYSTEM

The body's immunological function is performed by the lymphoid organs, including the spleen, thymus, tonsils, Peyer's patches located in the small intestine, bone marrow, and lymph nodes (**FIGURE 5-1**).

FIGURE 5-1

Anatomy of the immune system

Spleen provides lymphocyte proliferation, immune surveillance, and immune response; removes old or damage platelets and blood cells from the blood; removes debris and foreign matter from the blood. It has other functions not related to the lymph system that are not discussed here.

Thymus secretes thymosin and thymopoietin, hormones that enable T-lymphocytes to become activated against specific pathogens; it does not directly fight the antigens.

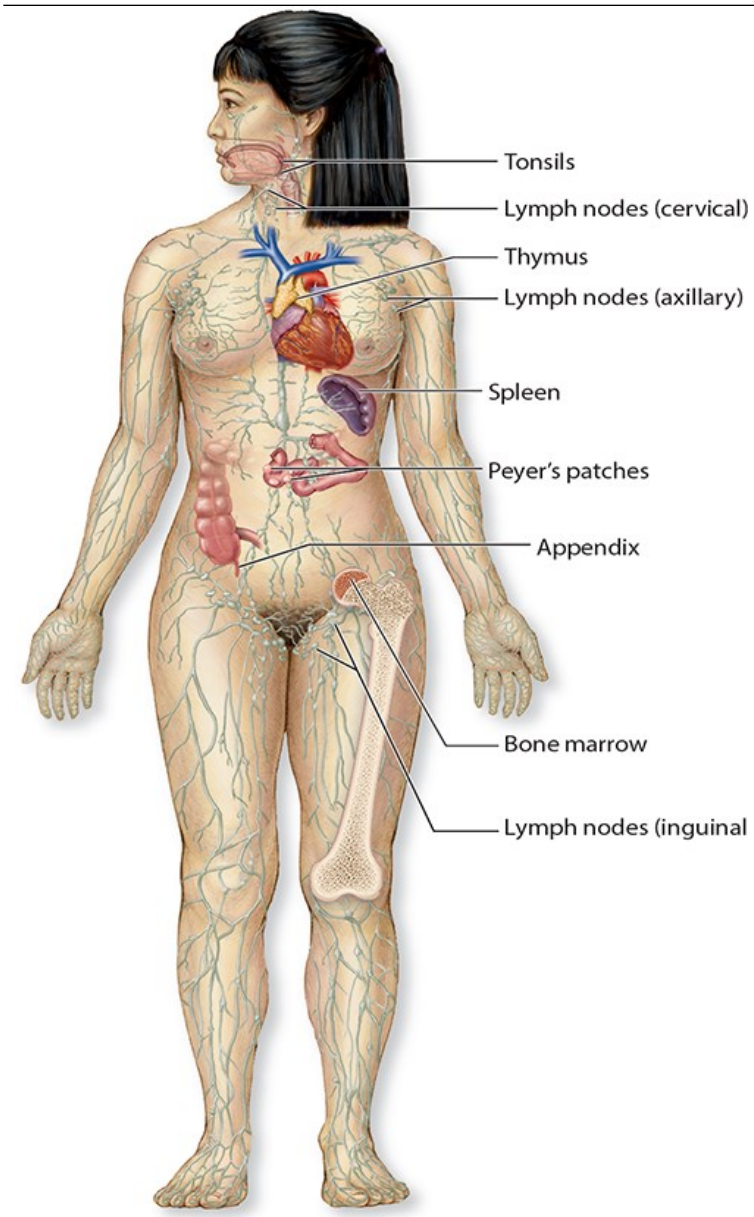
Tonsils capture and remove pathogens in the airway during inhalation.

Peyer's patches, structurally similar to tonsils, are located in small intestines. They destroy bacteria in the intestines and generate “memory lymphocytes” for long-term immunity.

Bone marrow, also called myeloid tissue, is responsible for production of all the cells of the immune system through the process of hemopoiesis. The stem cells become common myeloid stem cells or common lymphoid stem cells that further differentiate into red blood cells, platelets, and white blood cells.

Lymph nodes are the biological filter station for antigens present in lymph fluid; responsible for purifying and draining lymph fluid.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



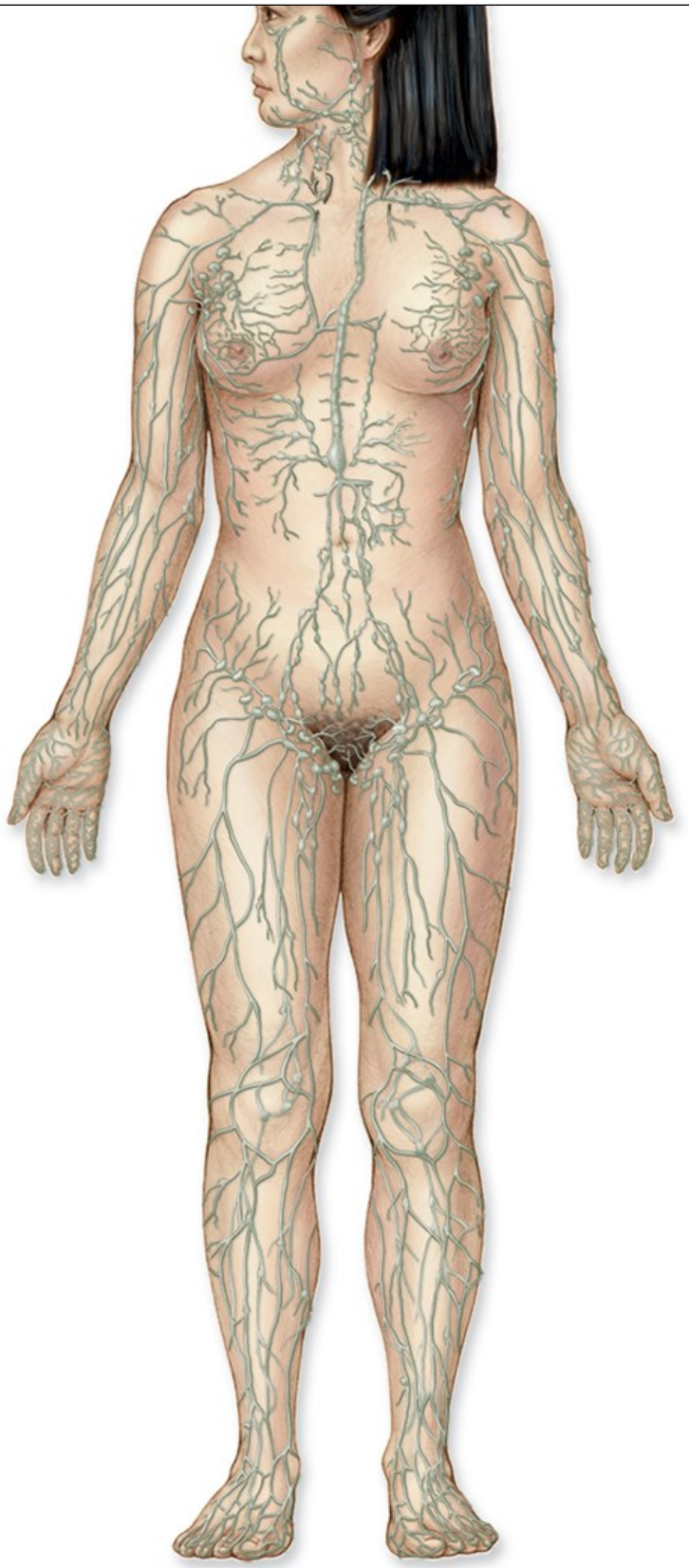
Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

The lymphatic system is a body-wide network of superficial and deep vessels connected by perforating vessels, similar to the venous system. It is comprised of lymph capillaries, lymphatic precollectors, collectors, ducts, and trunks (**FIGURE 5-2**). The superficial vessels, located directly under the skin and above the fascia, drain the dermis and subcutaneous tissues. The deep vessels are located below the fascia and drain all the tissue deep to the fascia. There are connecting vessels that perforate the fascia to transport the fluid from the deep vessel system to the superficial vessel system, which is the opposite of how the venous system functions. The lymphatic vessels are similar to arteries in that the larger collecting vessels have smooth muscle in the wall, and they are similar to the veins in that the collecting vessels contain valves that allow for unidirectional flow of the fluids.

FIGURE 5-2

Full-body lymphatic system The superficial network of lymphatic vessels have specific drainage routes to regional lymph nodes.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

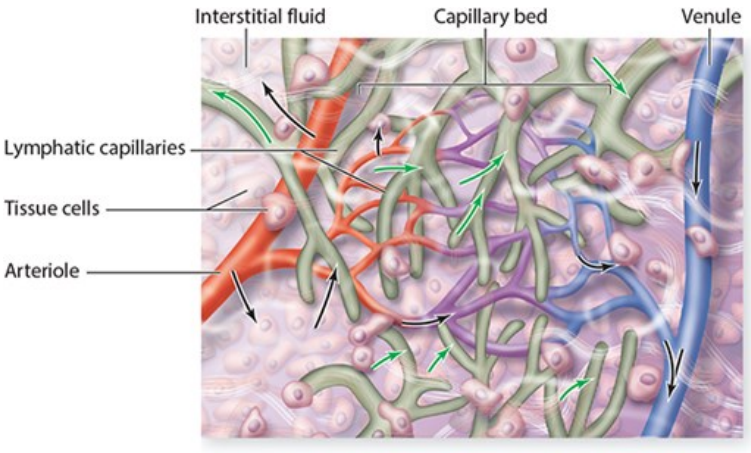
The superficial system begins with the lymphatic *capillaries* that appear much like a fishnet underneath all of the skin (**FIGURE 5-3**). The capillaries are

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

the smallest-diameter vessels and thus have the largest numbers. As the diameter of the more proximal lymphatic vessels increases, the number of lymphatic vessels decreases. Lymph capillaries have anchoring filaments that attach to the basement membrane between the dermis and epidermis (**FIGURE 5-4**). The lymph capillaries are more permeable than the vascular capillaries and therefore can absorb larger molecules of protein and fat. Each lymph capillary is composed of a single layer of overlapping flat endothelial cells, and the overlapping junction is called the *inlet junction*. The capillary does not contain valves, thus allowing the fluid to move in any direction within the capillary system.

FIGURE 5-3

Lymphatic capillaries that intertwine with the arterial and venous capillary bed Molecules and fluid flow from the arterial capillary bed into the tissue spaces (black arrows) and from the interstitial spaces into the initial lymphatic capillary, termed *reabsorption* (green arrows). Lymph fluid then flows from the initial lymph capillaries into slightly larger vessels, termed *precollectors*. Fat and protein molecules that are too large to enter the venous capillaries must be reabsorbed by the initial lymphatics or initial lymph capillaries.

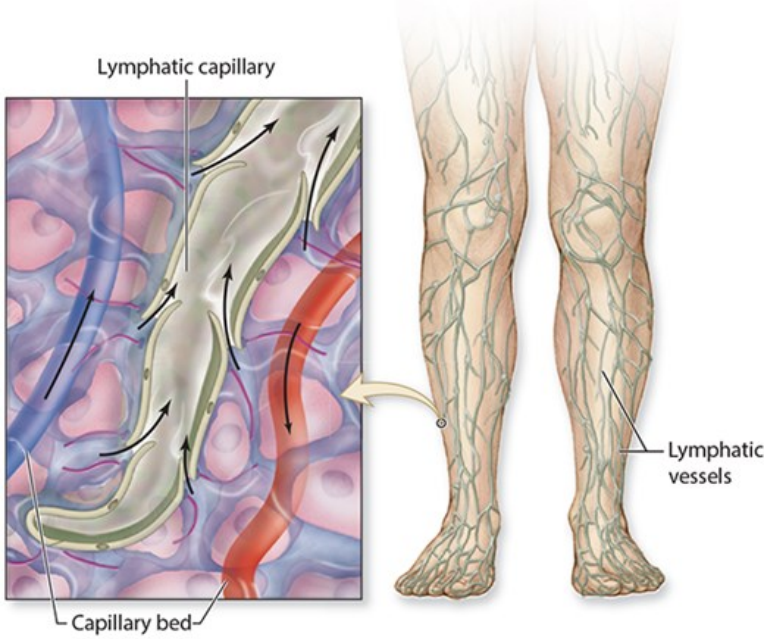


Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-4

Anchoring filaments of the lymphatic capillaries The initial lymphatic capillary is connected to the basement membrane by anchoring filaments. When the interstitial pressure gradient (ie, concentration of protein molecules) is high, tension is exerted on the anchoring filaments, which results in pulling the single layer of overlapping endothelial cells. This tension opens the filament and allows the fluid to enter the initial lymphatic capillary where the pressure is low.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

The capillaries merge and become *precollectors*, which have a larger diameter and therefore a lower pressure, thus facilitating movement of the fluid from the capillary to the precollector. The precollectors also have the overlapping endothelial anatomy that allows them to absorb interstitial fluid, and they have both a limited number of valves and a limited smooth muscle structure that allows them to initiate transportation of the collected fluids.

All of the precollectors merge to become larger diameter vessels, termed *collectors.* The collectors first bring the lymph fluid to the lymph nodes and then carry the remaining fluid to the larger *lymphatic trunks*. The anatomical structure of the collectors is similar to the construction of the veins in that they have three distinct layers (intima, media, and adventitia). They have well-defined valves that permit flow to go in only one direction—toward the heart. Within the lymph collector, the vessel segment between two valves is termed the lymphangion (**FIGURE 5-5**). Each lymphangion has an autonomic-driven resting contraction rate of approximately 10–12 contractions per minute.[1] This is further discussed under physiology. The superficial lymphatic collectors are located in the subcutaneous fatty hypodermis and they follow a direct route toward the regional lymph nodes. The deep lymphatic collectors follow the pathway of larger blood vessels. Lymph collectors drain lymph fluid from specific areas of the body, creating lymphatic "territories" that drain to specific regional lymph nodes. These lymphatic territories are defined by lymphatic watersheds that tend to delineate the body into specific collection and drainage patterns (**FIGURE 5-6**). Although there is some cross-connection between the territories via the watersheds, most of the fluid flows directly to its own regional lymph nodes. If there is fluid overload in one area, some of the fluid can be diverted to another region via the watershed connections.

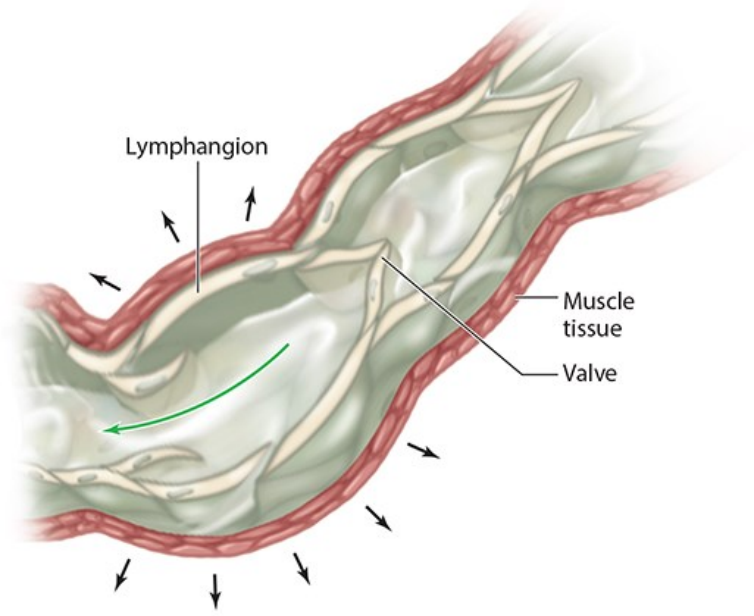
CLINICAL CONSIDERATION

The removal of any regional lymph nodes results in a reduced ability for the immune system to respond to foreign pathogens and an increased risk of developing infection.

FIGURE 5-5

Lymphangion The lymph vessels containing lymphangions are adjacent to the muscle; thus, when the muscle contracts the lymph flow is increased. Valves and smooth muscle in the angion promote directional flow of the fluid and prevent reflux, much like the valves in veins.

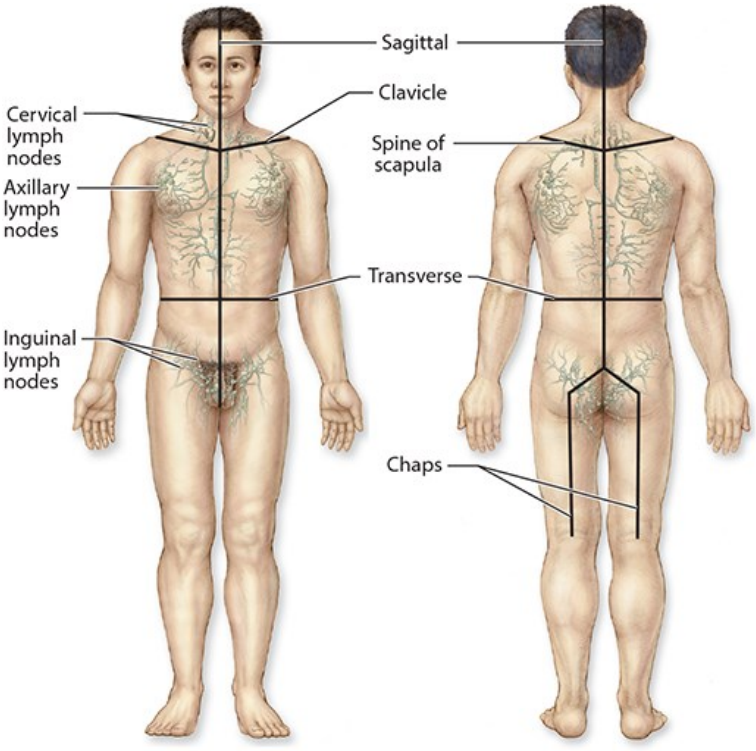
Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-6

Lymphatic watersheds The body is divided into lymph territories that allow drainage of the lymph flow from specific body regions to specific regional lymph nodes. The deep vessels do not cross between watersheds (areas of collection); however, there are some superficial vessels that cross the watershed boundaries and thereby divert lymph fluid from one quadrant to another when there are conditions of overload. The superficial pathways that cross between watersheds can be encouraged with manual lymphatic mobilization to direct more flow to open areas; however, it is usually inadequate to remove the majority of the fluid in the affected quadrant.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

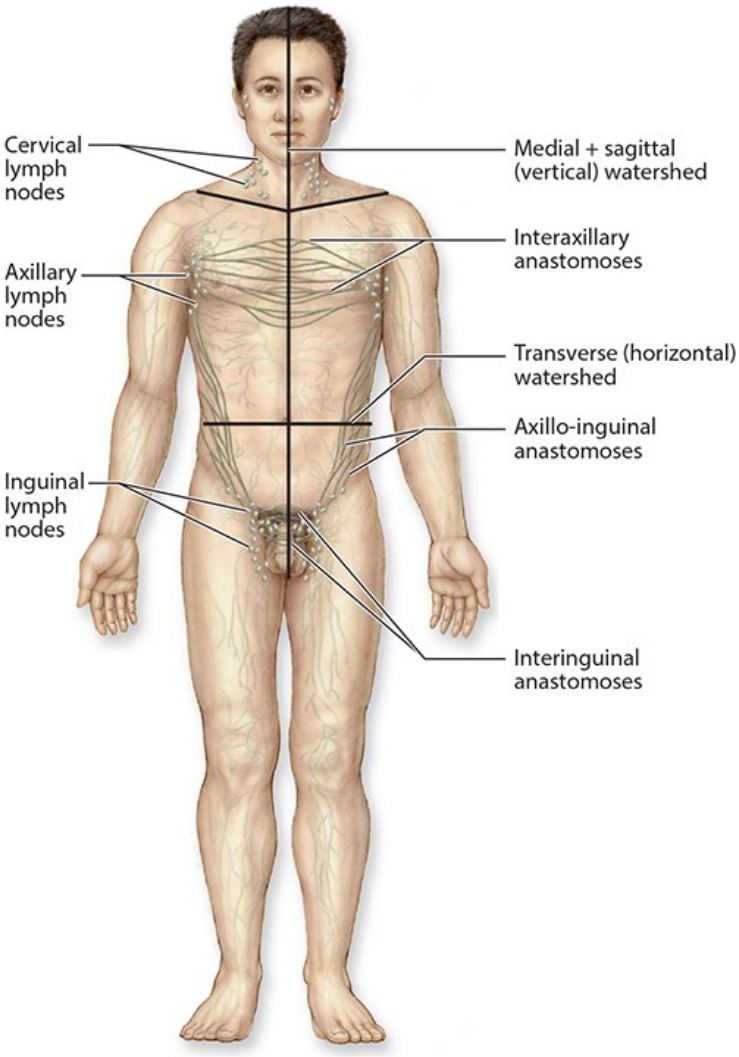
The lymph nodes (estimated to be about 700) are located in clusters throughout the body (**FIGURE 5-7**). The lymph node is bean-shaped and surrounded by a dense fibrous capsule (**FIGURE 5-8**). The collectors converge at the convex side of the lymph nodes where they are termed *afferent lymph vessels*. The fluid moves through the cortex of the node, into the medulla, and exits on the concave side via *efferent vessels* that then become trunks. The number of efferent vessels is less than the number of afferent vessels, resulting in a decreased transportation rate through the lymph nodes, and thus allowing the immune system cells time to phagocytose the pathogens that are in the lymphatic fluid.

CLINICAL CONSIDERATION

Lymph nodes become enlarged when they try to contain and destroy pathogens such as bacteria or cancer cells.

FIGURE 5-7

Clusters of lymph nodes throughout the body Lymph nodes are located strategically throughout the body, in conjunction with the superficial anastomoses that allow fluid to cross the watershed boundaries. The lymph system naturally diverts fluid across the watershed during times of overload; however, in lymphedema it is insufficient to manage the total volume of fluid needing to be transported. Manual lymphatic mobilization stimulates these anastomoses to increase the rate of flow.

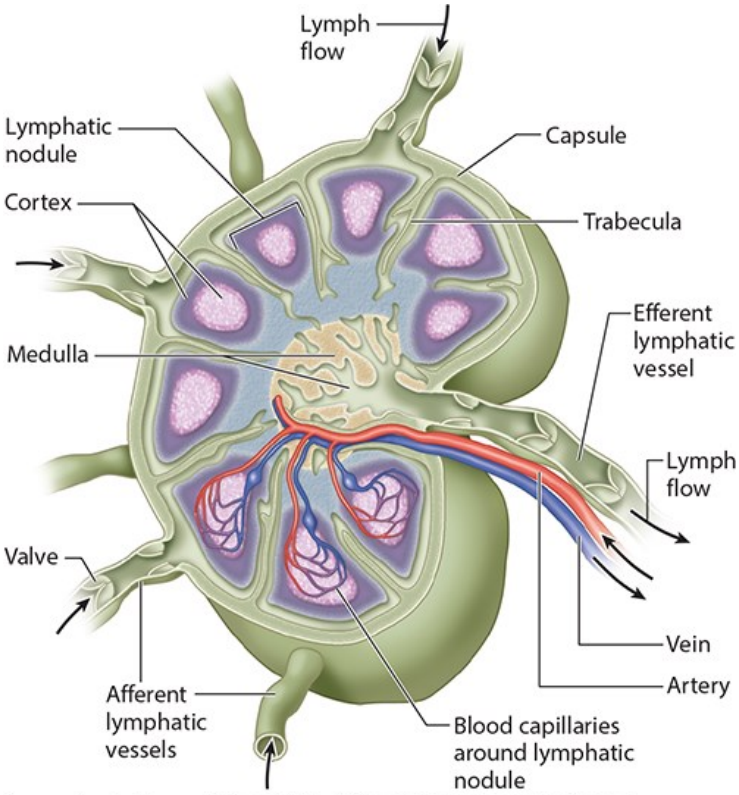


Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

FIGURE 5-8

Lymph node anatomy A greater number of afferent vessels bring fluid to a lymph node and a lesser number of efferent vessels leave the lymph node. This anatomical arrangement permits a slower rate of lymph transport through the lymph nodes and thus allows time for the immune system to phagocytose bacteria, waste products, and dead cells. For individuals with cancer who undergo a lymph node dissection, the rate of flow through the regional lymph node is automatically decreased. This sets the stage for potential lymphatic congestion in the affected quadrant and may lead to lymphedema.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

When the superficial and deep collectors converge, the vessels are termed *trunks.* The trunks are similar in construction to the collectors except they are larger in diameter and contain more smooth muscle in the vessel wall (**FIGURE 5-9**). The trunks are named as follows: right and left lumbar trunks, gastrointestinal trunk, jugular trunk, supraclavicular trunk, subclavian trunk, peristernal trunk, and bronchial-mediastinal trunk. The right and left trunks and the gastrointestinal trunk converge in the anterior area of T-11-L2 to form the *cisterna chyli*, which is the beginning of the thoracic duct. The thoracic duct ascends cephalically along the anterior vertebral column and perforates the diaphragm.

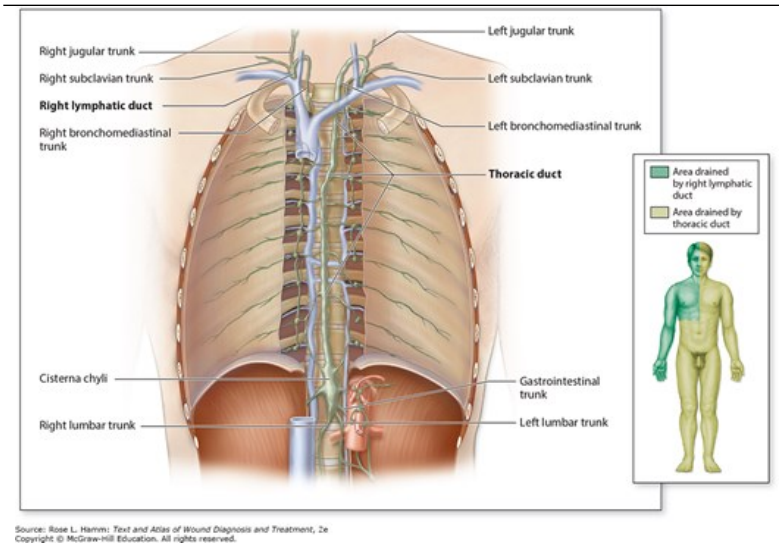
CLINICAL CONSIDERATION

Diaphragmatic breathing provides a force to promote lymphatic drainage from the thoracic duct into the venous angle. This results in a negative pressure gradient within the thoracic duct, therefore promoting distal-to-proximal lymph transport.

FIGURE 5-9

Lymphatic trunks and the territories that drain into the trunks The collecting vessels merge to form lymphatic trunks.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

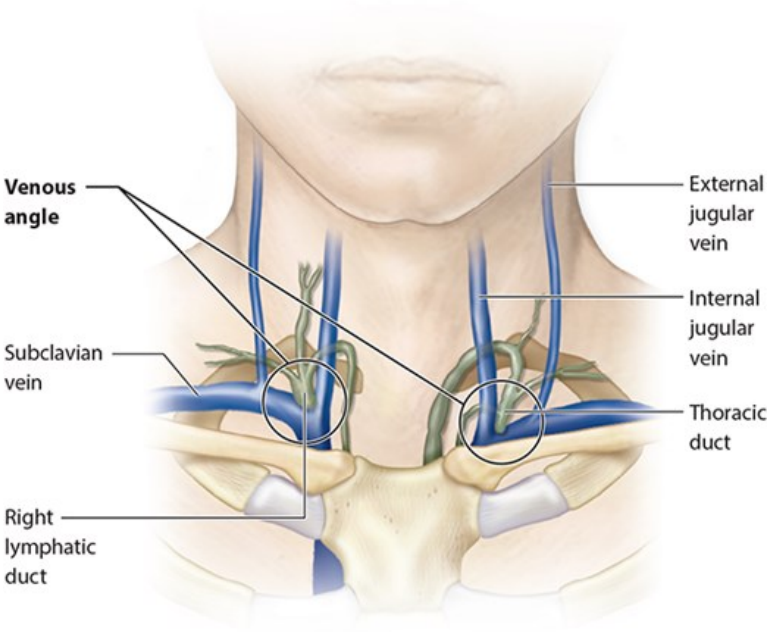


Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

The only direct connection between the lymphatic system and the venous system occurs where the thoracic duct (on the left) and the right lymphatic duct enter the venous angles. The venous angle is formed by the convergence of the internal jugular and subclavian veins (**FIGURE 5-10**). This is the anatomical termination of what is described as the lymphatic system.

FIGURE 5-10

Venous angle The venous angle is formed by the junction of the internal jugular vein and the subclavian vein. The thoracic duct on the left and the right lymphatic duct insert directly into this junction, which is termed the ***venous angle***. The venous angle flows into the brachiocephalic vein.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

PHYSIOLOGY OF LYMPHATIC FLOW

The purpose of the lymphatic capillaries is to absorb interstitial fluid and molecules, and when the fluid enters the capillaries, it is termed *lymphatic fluid*. The endothelial cell junctions overlap, creating inlet valves, so that when the interstitial protein concentration increases, tension on the anchoring filaments causes the inlet valves to open and allows absorption of the larger fat and protein molecules. These inlet valves remain open as long as the interstitial pressure (a result of plasma protein concentration) remains higher than the pressure within the lymph capillary.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

CASE STUDY

INTRODUCTION

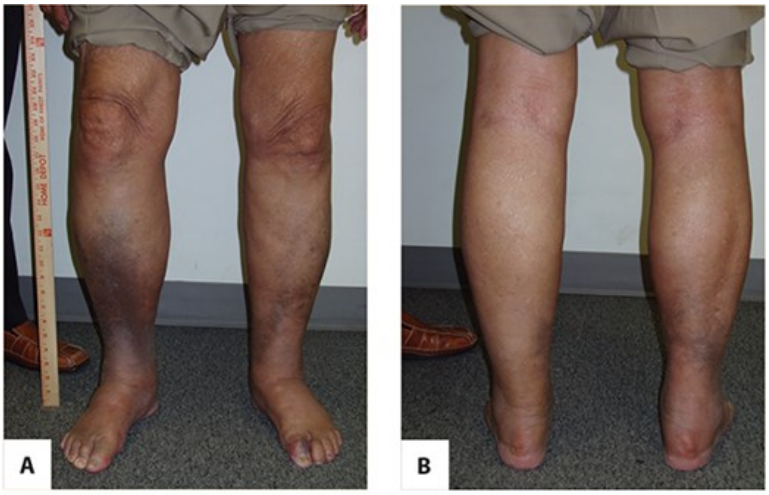
Mr. CB is an 80-year-old male who presents with bilateral lower extremity edema (left greater than right) that has recently worsened and is preventing him from playing golf. Past medical history includes history of myocardial infarct with residual cardiomyopathy (15 years ago), chronic renal insufficiency, Type 2 diabetes for which the patient takes oral medications, history of gout in the left great toe, and history of prostate cancer that was diagnosed 6 years ago and is being treated by "watchful waiting," meaning there is no treatment except yearly monitoring of PSA levels (**FIGURE 5-11A, B**).

DISCUSSION QUESTIONS

1. What questions are appropriate to ask the patient in order to obtain the subjective history?
2. What additional objective information is needed to determine the cause of the edema?
3. What tests and measures are indicated to determine the plan of care for this patient?

FIGURE 5-11

A, B. Case study photographs The case study patient presents with bilateral arterial, venous, and lymphatic insufficiency.



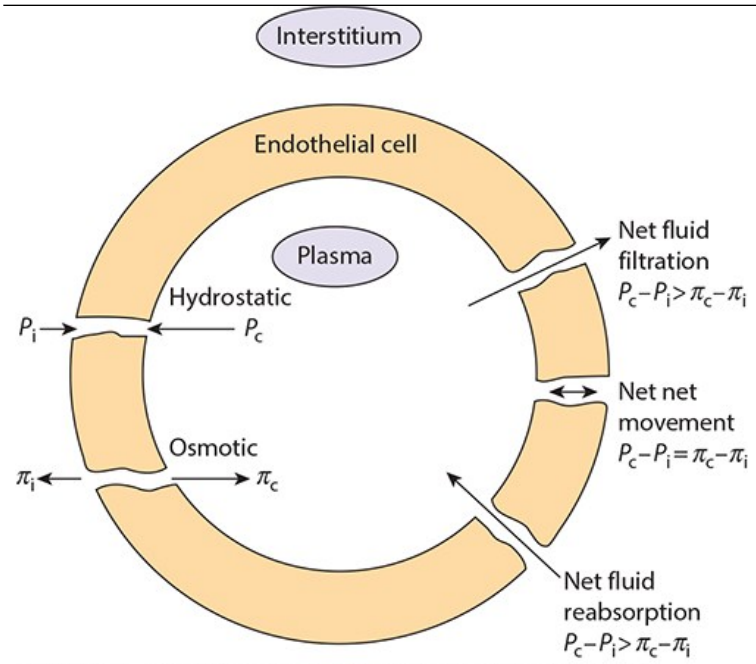
Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Fluid movement between the capillaries (arterial, venous, and lymphatic) and the interstitial spaces is governed by Starling's Law of Equilibrium. This law describes how the osmotic and hydrostatic pressures in the capillaries and interstitial tissue determine the direction of fluid movement. The irony of this equation is that equilibrium is never really achieved because of the dynamic nature of the human body, which does not allow the pressure gradient to equal zero; therefore, fluid is always moving, or in physiologic terms, fluid is constantly being filtrated and reabsorbed at the capillary bed (**FIGURE 5-12**).[2,3]

FIGURE 5-12

Starling's Law of Equilibrium

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

The difference between the net hydrostatic pressure and the net osmotic pressure is the force that determines the direction of fluid flow (**TABLE 5-2**). When the fluid flows from the interstitial space into the capillaries, it is termed *reabsorption* and can occur at both the venule and the lymphatic capillaries. When the fluid flows from the capillaries into the interstitial space, it is termed *filtration*. Equilibrium occurs when an equal amount of fluid moves from the arterial capillaries into the interstitial space and out of the interstitial space into the venous and lymphatic capillaries. Hydrostatic pressure is the pressure exerted by the blood on the capillary walls. Because the pressure is higher on the arterial side than on the venule side, fluid is pushed out of the capillary bed into the interstitial space. Osmotic pressure is the force created by large molecules such as plasma proteins, and it opposes hydrostatic pressure. Plasma protein molecules attract water, so that in a normal system they facilitate reabsorption of the fluids back into the venule.[4]

TABLE 5-2

Definitions for the Physiological Components of Starling's Law of Equilibrium

Capillary bed forces—Forces responsible for direction of fluid movement at the capillary bed membrane. There are 4 forces: 2 hydrostatic and 2 oncotic pressures that affect trans-capillary fluid exchange at the capillary bed.
Hydrostatic pressure (HP)—Pressure exerted on the vessel wall by the fluid (water).
Capillary hydrostatic pressure (HP_c)—Pressure from water that drives fluid out of the capillary (ie, filtration); is highest at the arteriolar end of the capillary and lowest at the venular end.
Interstitial hydrostatic pressure (HP_i)—Pressure generated by the volume of water in the interstitial spaces. As more fluid filters into the interstitium, both the volume of the interstitial space and the hydrostatic pressure within that space increase.
Net hydrostatic pressure (net HP)—Difference between the capillary HP (HP_C) and the interstitial HP (HP_{if}).
Osmotic pressure (OP)—Pressure exerted by plasma protein molecule concentration that is required to prevent the flow of water across a semipermeable membrane via osmosis.
Capillary osmotic pressure (OP_c)—Pressure that drives fluid into the capillary bed. The capillary membrane is readily permeable to ions; therefore, the osmotic pressure within the capillary is determined by the presence of plasma proteins (that are relatively impermeable to the capillary membrane). This

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

pressure is referred to as the “oncotic” pressure or “colloid osmotic” pressure because it is generated by colloids. Albumin generates about 70% of the oncotic pressure, which is typically 25–30 mmHg.

Interstitial osmotic pressure (OP_i)—Pressure of the interstitial fluid that depends on the interstitial plasma protein concentration and the reflection coefficient of the capillary wall. The more permeable the capillary barrier is to proteins, the higher the interstitial oncotic pressure. This pressure is also determined by the amount of fluid filtration into the interstitium.

Reabsorption—Process by which fluid moves from the interstitial spaces into the capillary bed.

Filtration—Process by which fluid moves from the capillary bed into the interstitial spaces.
Starling's hypothesis states that “the fluid movement due to filtration across the wall of a capillary is dependent on the balance between the hydrostatic pressure gradient and the oncotic pressure gradient across the capillary.”
The four Starling's forces are:

1. Hydrostatic pressure in the capillary (Pc)
2. Hydrostatic pressure in the interstitium (Pi)
3. Oncotic pressure in the capillary (pc)
4. Oncotic pressure in the interstitium (pi)

The balance of these forces allows calculation of the net driving pressure for filtration.
Under normal physiological conditions at the capillary bed, the exchange of fluids, nutrients, cellular debris, and waste products is primarily due to diffusion and filtration between the blood and tissues. Net filtration of fluid across the capillary membrane is governed by the balance of filtration forces and reabsorption forces. The direction of fluid movement is dependent upon the difference between hydrostatic pressure (HP) and osmotic pressure (OP). HP is the same as capillary blood pressure, which varies along the length of capillary, and under normal circumstances that pressure is lower in the venous end and higher in the capillary end.

Fluid is forced OUT of arterial end of capillary bed when:
Net hydrostatic pressure (net HP) = $HP_c - HP_{if}$ > Net osmotic pressure (net OP)$OP_c - OP_{if}$

FLUID IS FORCED OUT = FILTRATION

Fluid is forced INTO the venous end of the capillary when:
Net hydrostatic pressure (net HP) = $HP_c - HP_{if}$ < Net osmotic pressure (net OP)$OP_c - OP_{if}$

FLUID IS FORCED IN = REABSORPTION

To determine the ***resultant*** amount of fluid movement across the entire capillary bed:
Net filtration pressure = difference between:

Net HP – Net OP (arterial end) and NET HP – NET OP (venous end)

If the resultant difference is **positive**, the fluid is moved into the interstitial spaces.
If the resultant difference is **negative**, the fluid is reabsorbed into the capillary bed.
The one component missing from Starling's Law of Equilibrium is the lymphatic capillary. If the lymphatic capillary is healthy and functioning normally, the lymphatic capillary will reabsorb the protein molecules that escape at the arterial end of the capillary bed. If this process is functioning, there is no net buildup of interstitial pressure. However, if there is lymphatic insufficiency or overload, the normally negative pressure gradient within the initial lymphatic becomes positive (overload of protein molecules), at which point the initial lymphatics can no longer absorb the protein molecules. The result is the accumulation of protein molecules within the interstitial spaces. This will alter both the HP_{if} and OP_{if} pressure gradients of the capillary bed. Protein molecules attract water and are too large to enter the venous capillary. With a positive pressure in the lymphatic capillary preventing reabsorption into the lymphatic system, the protein molecules plus water accumulate in the interstitial spaces and eventually cause the skin to stretch, resulting in visible edema. Filtration is normally not affected.

The blood capillary wall is a semipermeable membrane, thus a force is required to move the larger protein molecules in and out of the capillary bed. At the arterial end where the net hydrostatic pressure is higher than the net osmotic pressure, the molecules are forced out of the capillary bed and into the

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

interstitium. However, at the venule end the net osmotic pressure is higher than the net hydrostatic pressure so the pressure is not sufficient to move the molecules from the interstitium into the capillary bed. Therefore, the molecules are reabsorbed into the lymphatic capillary at the overlapping endothelial junction. When this process is in balance so that fluids are adequately moved, no edema occurs. When there is an imbalance, for a variety of reasons and in either direction, edema develops. All edema pathology can be related to a problem with either filtration or reabsorption. When this happens, there is a transportation dysfunction—the freeway is clogged. The challenge to the clinician is to determine the pathology that prevents adequate flow of the fluid back into the central vascular system.

PATHOPHYSIOLOGY OF LYMPHEDEMA

Primary lymphedema is a result of congenital malformations of the lymphatic system, although the consequences may not be observed in the early years (**FIGURES 5-13** and **5-14**). *Secondary lymphedema* is a result of acquired damage to the lymph vessels or nodes and subsequent impaired reabsorption and/or transportation of lymphatic fluid (**FIGURE 5-15**). Refer to **TABLE 5-1** for more details of both types.

FIGURE 5-13

Primary lymphedema Primary lymphedema in an Advanced Stage II progressing to Stage III; presents with significant skin changes and open wounds on L leg.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-14

A. Lymphedema praecox (anterior view). B. Lymphedema praecox (posterior view) Anterior and posterior views of Stage II primary lymphedema as a result of cancer and its treatment. Note that the contour of the left lower extremity is proportionate even though the size discrepancy is obvious.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-15

Secondary lymphedema Stage II secondary lymphedema as a result of cancer and its treatment that resulted in clogged lymph nodes.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

A frequent cause of secondary lymphedema is chronic venous insufficiency (**FIGURE 5-16**).[5,6] The sequence of events is as follows: The venous system becomes compromised and cannot accommodate the normal amount of venous flow. The lymphatic system, when healthy, can compensate because it

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

can increase its rate of lymphangion contraction up to 10 times its resting volume and transport the fluid normally carried by the venous system.[1] For edema to develop in patients with chronic venous insufficiency, one of two scenarios must occur. *Either the lymphatic and venous load exceeds the maximum transport capacity of the lymphatic system or the lymphatic system becomes impaired and cannot transport the usual fluid load.* The lymphatic impairment may be caused by incompetent lymphatic valves due to distended vessels.[7,8]

FIGURE 5-16

Chronic venous insufficiency with secondary lymphedema Secondary lymphedema is frequently a consequence of chronic venous insufficiency. The hemosiderin stains (a byproduct of autolysis of hemoglobin released from red blood cells trapped in the interstitial tissue) are characteristic of chronic venous insufficiency. The pedal edema on the dorsum of the foot is also characteristic of secondary lymphedema.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment, 2e*
Copyright © McGraw-Hill Education. All rights reserved.

In the United States, the most recognized cause of lymphedema is related to cancer and its treatment (**FIGURE 5-17**).[9] The location and the severity of oncology-related lymphedema are influenced by the area affected, the location and total dosage of radiation, the extent of lymph node dissection, obesity, and presence of co-morbidities.[10,11] Clinical presentation can vary drastically and treatment has to be individualized. Other causes of secondary lymphedema include trauma, infection, surgery in which the lymphatic vessels are severed or damaged, and systemic disorders (eg, liver, kidney, or cardiac disease) (**FIGURES 5-18** to **5-20**). However, any pathology that results in damage to the lymphatic system with decreased transport capacity will result in chronic secondary lymphedema, and clinical presentation will be essentially the same with variations only in relation to the severity of the damage.

FIGURE 5-17

Lymphedema as a result of cancer and radiation (head and neck cancer) Lymphedema as a result of radiation can occur in any body part where the lymphatic vessels are damaged and the flow is interrupted. Note the changes in the skin, termed *dry desquamation*, in which the epidermal layer is sloughing much like superficial sunburn.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-18

Secondary lymphedema due to trauma The tight, orange-peel texture of the lower leg skin is a result of lymphedema that has developed after a lateral ankle injury. Any trauma that disrupts the lymphatic flow, limits joint range of motion, and alters gait cycle can result in lymphedema formation.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-19

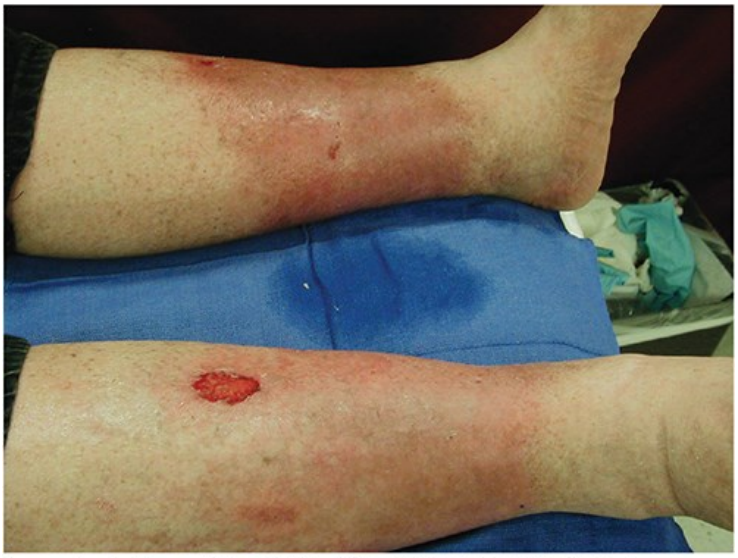
Secondary lymphedema due to orthopedic surgery Secondary lymphedema is common after lower extremity surgeries that interfere with lymphatic flow through the popliteal or inguinal lymph nodes, such as total knee or total hip replacements. Early mobilization, isometric exercises, and compression with short stretch bandages are recommended to prevent or treat this type of edema. Reduction of the edema through these strategies will subsequently result in better functional outcomes.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-20

Secondary lymphedema due to systemic disorder Bilateral lymphedema may be indicative of a systemic disorder (eg, congestive heart failure as in the case of this patient). The erythema of both lower legs is suggestive of cellulitis, which would be treated with antibiotics. Compression with short stretch bandages is recommended after both the congestive heart failure and the cellulitis have been treated.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

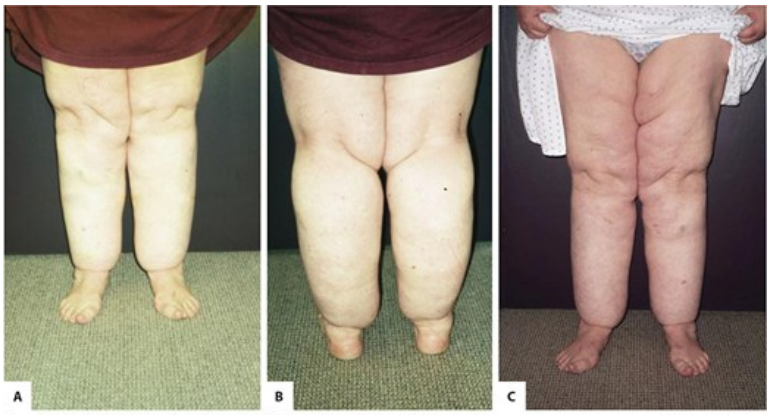
Lipedema (**FIGURE 5-21**) is a chronic disease of lipid metabolism that is often misdiagnosed as lymphedema.[12] Although the clinical presentation is similar, there are distinct differences that aid in making a differential diagnosis. For example, there is no edema in the feet, the edema is either bilateral or quadrilateral, the texture is soft and non-pitting, the skin temperature may be cooler than the trunk, and the skin may be hypersensitive to light touch. The underlying pathology is thought to be due to an abnormal deposition of adipose cells in the subcutaneous tissue, as well as a possible inheritance of genetic X-linked abnormalities.[13] Over time, however, if the superficial lymphatic system becomes compressed by the adipose tissue, secondary lymphedema can develop and the feet will also become edematous and pitting edema can occur.[14]

FIGURE 5-21

Lipedema **A** and **B** at the time of initial evaluation and **C** is after 4 months of treatment. Lipedema is often mistaken for lymphedema. Note the columnar

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

shape of the legs, sparing of the feet and ankles, and the significant skin folds above the ankles. Lipedema is a hereditary disorder and ***not*** a result of obesity.



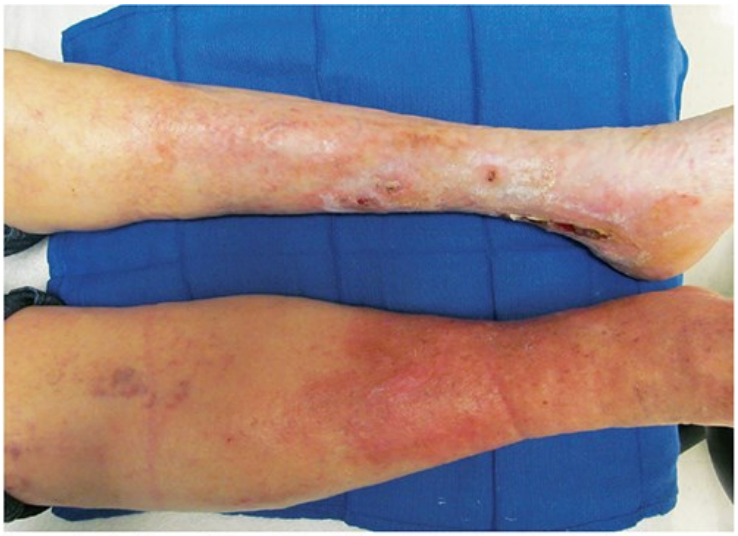
Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Stages of Lymphedema

The International Society of Lymphology has identified and defined the stages of lymphedema, as described in **TABLE 5-3**.[15] Because it is a chronic inflammatory condition, over time there is deposition of collagen fibers and adipose cells in the interstitial tissue of the affected region. Once the diagnosis of lymphedema is established, the severity is used to determine the stage of the disease. The extent of fibrosis and adipose is defined in the stages and is used to determine the patient prognosis (**FIGURE 5-22**).

FIGURE 5-22

Lymphedema as a result of occlusion to the abdominal lymph nodes Sudden onset of unilateral edema that is hard and does not respond to compression is suggestive of pelvic or abdominal lymph node obstruction. This patient was being seen for wounds on the left lower extremity; however, the severe, sudden-onset edema of the right leg was the result of a rare abdominal malignancy that caused node obstruction.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-3

Stages of Lymphedema

Stage 0	Stage 1	Stage 2	Stage 3
Subclinical stage: Lymph transport is Impaired; however, lymphatic load has not yet exceeded the transport capacity. Swelling is not evident. Clinical symptoms: Patients may report heaviness, tightness, or sensory changes in the affected limbs. This stage may persist for months or years before edema is detectable.	Edema may be variable, ie, decrease with rest or sleep and increase with activity Edema is very soft, easily pits with little to no fibrosis. Elevation of the limb results in dissipation of the edema. Patient may report heaviness, tightness, sensory changes, or pain in the limb.	Edema no longer dissipates with elevation of the limb. Palpation is positive for fibrosis and results in indentation of the skin. Fibrosis ranges from soft and pitting (early stage II) to hard and non-pitting (late stage II). Both are commonly present in various locations along the involved limb. Skin mobility may be decreased and texture may be brawny. Positive Stemmer sign • UE: may be present in the dorsum of the fingers. • LE is commonly present in the dorsum of the toes. Increased likelihood of skin infection.	Palpation: The fibrotic changes of the skin and subcutaneous tissues continue to increase in severity. Fat deposits in the subcutaneous tissues lead to the following clinical signs and symptoms. Little to no skin mobility is present. Hair follicles may be coarse or absent. Subcutaneous fat deposits and worsening of fibrosis result in increased skin folds, loss of joint spaces, papillomas, skin hyperpigmentation, hyperkeratosis, cysts, and fistulas. Pitting is absent or minimally present with significant pressure and, if present, dissipates rapidly. Palpation of the limb is hard. Stemmer sign intensifies. Patient has history of recurrent bacterial infections/fungal infections. Volume of lymphedema significantly increases resulting in loss of joint spaces and bony prominences, and limb shape is unrecognizable.

Risk Factors for Lymphedema

Risk factors for lymphedema formation that have been identified are listed in **TABLE 5-4**. In addition, recent research suggests that certain individuals may have a genetic predisposition to lymphedema development; this is especially important for the oncology population.[16] Evaluation of a patient with peripheral edema needs to include investigation of any comorbidities that may impair the lymphatic and venous systems. The development of secondary lymphedema is typically a result of multiple risk factors and/or insults to the lymphatic system; therefore, a complete systems review is required for any patient who presents with peripheral edema.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-4

Risk Factors for Secondary Lymphedema

UE Lymphedema	LE Lymphedema
History of UE venous pathologies	History of LE venous pathologies
Surgical damage or removal of axillary lymph nodes/vessels	Surgical damage or removal of pelvic, abdominal, or inguinal lymph nodes/vessels
	Multiple pregnancies
	Abdominal surgery
	Kidney, liver, cardiac dysfunction, or failure
	Hypertension
Radiation therapy, fibrosis	Radiation therapy, fibrosis
Genetic predisposition	Genetic predisposition
Comorbidities: autoimmune diseases, obesity	Comorbidities: autoimmune diseases, HTN, obesity, DM
Infections, seroma, wounds	Infections, seroma, wounds
Smoking	Smoking
Scar tissue	Scar tissue

Any pathology that compromises lymphatic reabsorption and transportation is a risk factor. Usually a combination of risk factors is the cause of lymphatic insufficiency and secondary lymphedema. The most commonly overlooked risk factor is chronic venous pathologies; the most recognized cause is oncology-related secondary lymphedema (ie, with breast cancer). In oncology-related secondary lymphedema, surgery and radiation therapy can either independently or combined result in secondary lymphedema.

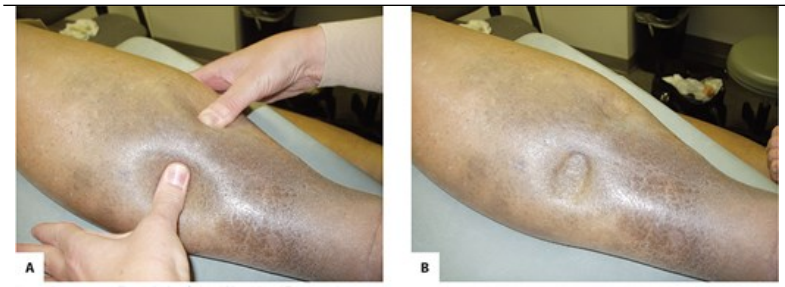
Clinical Presentation of Lymphedema

Evaluation of the clinical presentation of lymphedema includes both the volume of the extremity that is involved and the degree of fibrosis in the interstitial tissue. In the early stages, the edema is soft and pitting (**FIGURE 5-23**); however, as more collagen fibers are deposited, the tissue becomes more firm and less compressible. As the fibrosis becomes more severe, the tissue becomes non-pitting. Both pitting and non-pitting edema are often present in the same extremity. Infection is one of the primary causes of progression of lymphedema to a more severe stage.

FIGURE 5-23

A, B. Pitting edema Failure of the tissue to rebound after pressure with a finger is termed *pitting edema* and occurs during the early stages of lymphedema. As a result of chronic lymphedema, the subcutaneous tissue becomes fibrotic and does not pit; this is a characteristic of later stages of lymphedema. During the earlier stages, the deeper the pitting and the longer the rebound time, the better the prognosis for resolution of the symptoms. If the tissue is very thick and hard, there will be little to no pitting and a fast refill time, both indicating that longer treatment time and more compression therapy will be needed to remodel the fibrosis.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Patient complaints in the early stages of lymphedema include heaviness, tightness, aching, and sensory changes.[17,18] Corresponding to these complaints, the evaluator will note a loss of skin mobility; as the lymphedema progresses, the skin becomes tighter and less mobile. The difficulty or inability to mobilize or "tent" the skin over the dorsum of the foot (metatarsals) is termed the *Stemmer sign* (**FIGURE 5-24**).[5,19,20] This tightness of the skin and underlying tissue may also result in loss of range of motion and subsequently functional impairments.

FIGURE 5-24

Stemmer sign Inability to tent or pinch the skin at the distal foot is termed a positive Stemmer sign and is indicative of decreased skin mobility and elasticity as a result of chronic lymphedema.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

The clinical presentation of lymphedema associated with malignancy, radiation, or surgical interventions may involve any affected body part (eg, breast, head and neck, tongue, genitals). Lymph node dissection or radiation scarring may cause decreased lymphatic transport to the regional lymph nodes and subsequent fluid accumulation (edema) in the quadrant or territory distal to the injured nodes. In addition, if there is compromise to the pelvic and abdominal lymph nodes and vessels, genital and/or lower extremity edema may develop. As the stages progress, the fat and fibrosis lead to loss of anatomical landmarks such as joint lines and skin folds (**FIGURE 5-25**). Skin changes that may occur include hyperkeratosis, papillomatosis, and lymphocoeles (**FIGURE 5-26**).

FIGURE 5-25

Anatomic changes in late stages of lymphedema Anatomic changes as a result of lymphedema include the loss of joint spaces, toe deformities, hyperkeratosis, papillomas, and color variations in the skin.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-26

Skin changes as a result of lymphedema Changes in the skin color and texture occur with chronic lymphedema, including thick skin, discoloration, papillomatosis, and in this case, scarring from recurrent wounds on the lateral leg. Note the fungus, termed *onychomycosis*, on the toe nails.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Differential Diagnosis of Lymphedema

The presence of any edema is a symptom of some underlying pathology that has to be identified in order to have an effective plan of care. Diagnosis is based upon the patient history, physical exam, and laboratory tests to confirm or rule out systemic pathologies (eg, endocrine, cardiopulmonary, renal, or hepatic diseases) or vascular pathologies (eg, deep vein thrombosis). Some of the questions that can help with the differential diagnosis process include the following:

- Is the edema unilateral or bilateral?
- Was the onset sudden or gradual?
- Is the edema local or widespread?

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

- What are the patient comorbidities?
- Is there a history of cancer?
- Is there a history of venous disorders?
- What are the medications? (See **TABLE 5-5**.)
- What are the psychosocial behaviors?
- What is the pain pattern?

Clinical guidelines for diagnoses of upper quadrant lymphedema secondary to cancer were developed by the Oncology Academy of the American Physical Therapy Association.[21] Specific guidelines include the following recommended assessment tools: self -report of symptoms, bioimpedance analysis, volume measurements calculated from circumferential measurements, and/or water displacement (**FIGURES 5-27** and **5-28**)[42]. Circumferential measurements are converted to a volume using the formula for a truncated cone (**FIGURE 5-29**).[22,43] This process, which can also be used for lower extremity lymphedema, is essential for ensuring accurate diagnosis and for measuring outcomes. Recommendations were developed based on the stage of the lymphedema and the body part involved.[21]

FIGURE 5-27

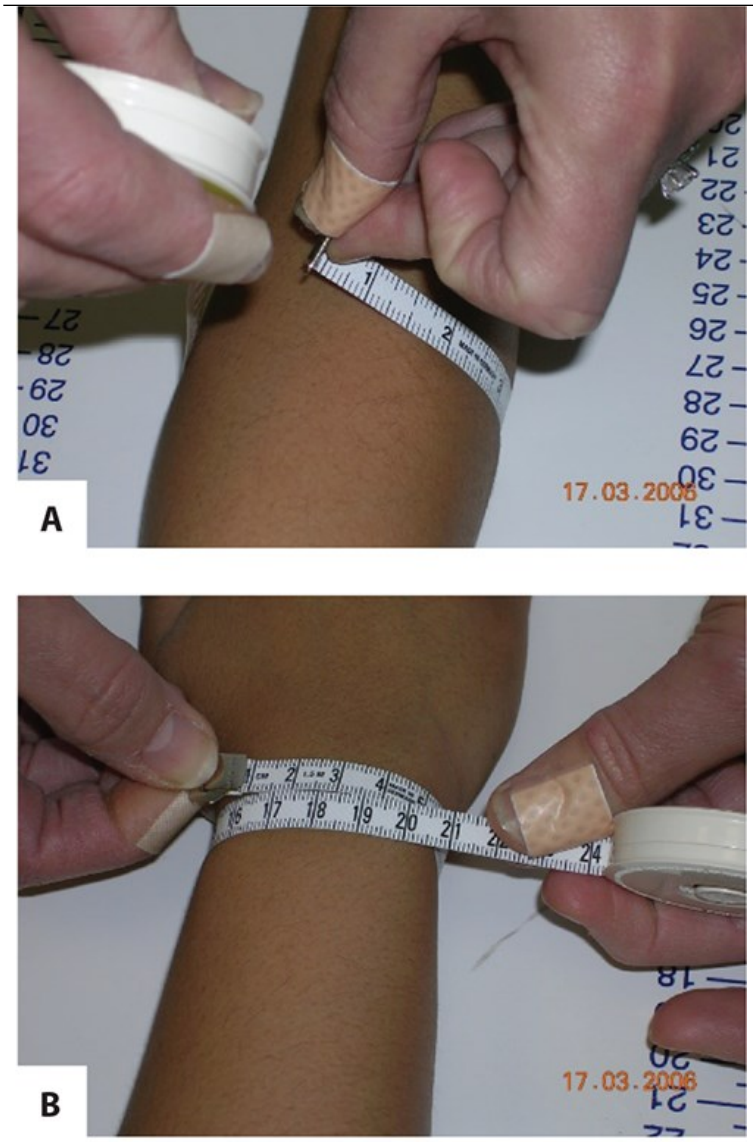
A, B. Volumetric measurement: water displacement technique A illustrates Equipment set-up to perform displacement volumetric measurements. When the extremity is inserted into the tank of water, the displaced water flows into the blue container; when the extremity is extracted, the amount of water displaced is measured (**B**).

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-28

A, B. Volumetric measurement: tape measure technique A and **B** demonstrate the upper extremity circumferential measurement technique. The tape is placed perpendicular to the arm, proper "tautness" is ensured by applying enough tension to take up the slack but not wrinkle the skin, and measurements are recorded at least two times and averaged for the results. (Photographs used with permission of Marisa Perdomo)

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-29

Summed Truncated Cone for the upper extremity.

Summed Truncated Cone
$V = (h)(C^2 + Cc + c^2)/12\pi$
The arm is divided into "segments" with the "h" being constant either 4 cm or 10 cm for the arm and typically 10 cm for the leg. Each of the segments is considered a "mini" cone and all of the segments are summed to determine the volume of the limb.

h = height of cone; C = proximal circumferential;
c = distal circumference

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-5

Medications That May Contribute to Peripheral Edema

Anti-arrhythmics (amiodarone, Norpace, Flecainide)
Anti-coagulants (Plavix, Crestor, Lipitor, Arixtra, Cilostazol)
Antidepressants
Antifungals (Amphotericin)
Antihypertensive (nitrates, hydralazine, minoxidil)
Anti-neoplastic (Xeloda, Aromasin, Herceptin, Zometa)
Anti-osteoporotic agents (Actonel, Bisphosphates)
Antisympathetics (reserpine, guanethidine)
Beta-blockers (Tenormin, Coreg, Labetalol, Toprol)
Calcium channel blockers (Verapamil, Nifedipine)
Chemotherapy agents (docetaxel, cisplatin, tretinoin)
Centrally acting agents (clonidine, methyldopa)
Corticosteroids
Direct vasodilators (hydralazine, minoxidil, diazoxide)
Glitazones, Actos, Avandia (DM)
Hormones (estrogens/progesterones/testosterone)
Nonsteroidal anti-inflammatory agents
Nonselective cyclooxygenase inhibitors
Selective cyclooxygenase-2 inhibitors
Troglitazone
Phenylbutazone
Parkinson agents (Mirapex)
Valproic acid

From Cho S, Atwood E. Peripheral edema. *Am J Med.* 2002;113:580–586, **Table 5-2**.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Several imaging techniques to assist in diagnosis, treatment planning, outcome measurements, and research have been discussed in the literature. *Radionuclide lymphoscintigraphy* involves subcutaneous or intradermal injection of a radiotracer (usually Tc filtered sulfur colloid with a particle size <100 nm) into the involved and the uninvolved extremity (for control data), followed by nuclear imaging.[23,24] Qualitative and quantitative readings can be used to confirm a differential diagnosis of lymphedema, assess the efficacy of therapeutic interventions, and to predict the risk of post-operative lymphedema.

Near-infrared (NIR) fluorescence imaging with indocyanine green is a "non-invasive assessment of both lymphatic architecture and function as well as potential disease markers of lymphatic function."[25] Clinical applications include intraoperative assessment of lymphatic vessel patency, delineation of tumors, and assessment of lymphatic flow both during and after therapeutic interventions.[26]

An additional aspect of patient evaluation is the effect of the lymphedema on quality of life. A systemic review of patient-reported outcome instruments regarding quality of life among breast cancer patients with lymphedema was reported by Pusic et al.; they recommended the *Upper Limb Lymphedema 27 (ULL-27)* as it demonstrated strong psychometric properties (**TABLE 5-6**).[27] The *Lymph-ICF-LL* for patients with lower extremity lymphedema consists of 28 questions for the following domains: physical function, mental function, general tasks/household activities, mobility activities, and life/social domain. Psychometric properties and test-retest reliability are both reported as acceptable to good range.[28] The *Patient Benefit Index (PBI)* allows patients to rate the extent to which their goals have been met and consists of the following two parts: (1) the Patient Needs Questionnaire in which patients rate the importance of 23 patient-relevant goals on a scale of 1–5 ("not at all" to "very") and (2) the Patient Benefit Questionnaire in which patients rate the extent of their goal achievement on the same 1–5 scale.[29] The Freiburg Life Quality Assessment for lymphedema (FLQA-L) consists of 92 items, and was used to develop and validate the FLQA-LS, which consists of 33 items. The shortened version demonstrated validity and feasibility for use in both clinical and research settings.[30] Regardless of the tool used, assessing health-related quality of life is an integral part of the total evaluation of patients with lymphedema.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-6

Upper Limb Lymphedema 27 (ULL-27) Quality of Life Scale

Physical functioning
Psychological dimension
1. Difficulties grasping high objects
2. Difficulties maintaining certain positions
3. Arm feels heavy
4. Arm feels swollen
5. Difficulties dressing
6. Difficulties getting to sleep
7. Difficulties sleeping
8. Difficulties grasping objects
9. Difficulties holding objects
10. Difficulties walking/heavy arm
11. Difficulties washing
12. Difficulties taking public transport
13. Tingling, burning feelings
14. Feelings of swollen, hard, tense skin
15. Difficulties in working relationships and tasks
Psychological dimension
16. Feeling sad
17. Feeling discouraged
18. Feeling a lack of self-confidence
19. Feeling distressed
20. Feeling well in one's self
21. Feeling a wish to be angry
22. Having confidence in the future
Social Dimension
23. Difficulty taking advantage of good weather, in life outside the house
24. Difficulty with personal projects, holidays, or hobbies
25. Difficulties in emotional life with spouse or partner
26. Difficulty in social life
27. Fearful of looking in a mirror

Adapted from Launois R, Mègnigbêto AC, Pocquet K, Alliot F. A specific quality of life scale in upper limb lymphedema: the ULL-27 questionnaire. In C. Campisi, MH Witte, CL Witte (eds). Progress in lymphology: XVIII International Congress of Lymphology—September 2001, Genoa (Italy). *Lymphology* 35(suppl):1–760; 2002:181–187. Available at: http://www.rees-france.com/en/IMG/pdf/ART-1002_ULL27__ISL_Genes_-2.pdf. Accessed July 23, 2018.

TREATMENT OF LYMPHEDEMA

Treatment of any disorder begins with effective prevention, and the best strategy for prevention of secondary lymphedema is to optimize lymphatic flow so that if the lymphatic system is overloaded (eg, with CVI) or damaged (eg, with surgery, trauma, or radiation), the transport capacity remains sufficient to manage the lymphatic load. This is especially important for patients who have chronic venous insufficiency in whom the lymphatic system must compensate for the venous insufficiency by increasing its transportation capacity (eg, by increasing lymphangion contraction rate) over a long period of time. The chronic high demand placed on the lymphatic system leads to valvular distention and a lower lymphangion contraction rate, resulting in

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

secondary lymphedema.[31] See **TABLE 5-7** for strategies to optimize both venous and lymphatic flow.

TABLE 5-7

Strategies to Optimize Venous and Lymphatic Flow

Exercise	Compression Therapy	Precautions
FOR LYMPHEDEMA:		
Diaphragmatic breathing. Range proximal joints first and move distally; begin with active and gradually add resistance. Aerobic exercise. End exercise session with diaphragmatic breathing.	Multilayer compression bandages with short-stretch bandages. Intermittent compression pumps. Garments to assist in maintaining reduction of lymphedema. Best used after physical therapy reduces the lymphedema.	Maintain healthy skin. Wear non-restrictive clothing. Avoid excessive weight gain.
FOR VENOUS INSUFFICIENCY WITHOUT LYMPHEDEMA:		
Ankle mobilizations and stretches. Ankle pumps. Heel/toe raises, sitting and progress to standing. Walking with exaggerated heel/toe sequence.	Multilayer compression systems. Semi-rigid compression with short stretch bandages. Compression stockings measured to fit the individual. Garments essential for prevention of venous dysfunction after DVT to prevent CVI. Intermittent compression pump as adjunct to self-management program.	Avoid prolonged sitting with legs crossed. Avoid prolonged standing. Wear non-restrictive clothing. Avoid smoking.

The first step in treating any peripheral edema is determining and treating any underlying pathologies that are contributing to the edema formation. Whereas compression has traditionally been recognized as the primary intervention for edema, it is now recognized that exercise, manual lymphatic mobilization, compression pumps, and skin hygiene are equally important for optimal outcomes.[32,33] The combination and sequence of therapies depend on the etiology and the individual presentation.

CLINICAL CONSIDERATION

A cardiovascular exercise routine is beneficial for both systems; however, the 24-hour response needs to be monitored and assessed if lymphedema increases, in which case the intensity or duration of exercise is decreased.

CASE STUDY

Subjective information obtained from the patient:

- Reports a mild heart attack and takes the following medications:
  - Lipitor, 20 mg
  - Coreg (beta blocker), 6.25 mg
  - Metoprolol, 100 mg daily

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

- Coumadin, varies with INR, from 2–4 mg daily
- Lasix, 40 mg daily
- Metformin hydrochloride, extended release, 1000 mg daily,
- ASA, 81 mg

- Reports his last ejection fraction was 45%; patient is seen by his cardiologist two times a year.
- Reports being careful regarding high-fat, high-cholesterol foods, but admits to liking ice cream daily.
- Works as owner of a moving van company and goes to office daily.
- Uses a rolling walker with a seat in all environments except at work.
- Lives with his wife in a one-story house with a daily housekeeper.

Objective information obtained from tests and measures:

- Unable to sit or stand without upper extremity assist; with UE assist, able to do 3 in 30 seconds.
- Six-minute walk test with the walker, 390 yards.
- Bilateral trendelenburg gait with the rolling walker, has little or no heel/toe sequence during gait cycle.
- ABI: left is 0.65, right is 0.7.
- Pulses: left DP 1+, PT 2+; right DP 2+, PT 2+.
- Auscultation of lungs—negative for crackles or congestion, decreased breath sounds in bilateral lower lobes.
- Auscultation of heart—negative for S3 heart sounds.
- Monofilament testing: + response to 5.07 monofilament on both plantar feet, all points.
- Range of motion—hip extension bilaterally to neutral, lacks 5° of knee extension bilaterally (5–120° knee flexion), 5° DF, 20 PF.
- Strength—gluteus medius bilaterally 3–/5, hip extensors 3–/5, ankle 2+/5.
- Patient reports glucose levels range from 120 to 200 fasting, last HbA1C (per lab results) was 7.5.

DISCUSSION QUESTIONS

1. What are the patient's risk factors for lymphedema?
2. What is the physiological rationale for his lymphedema?
3. Is there any other information needed before treatment can be initiated?

Before compression can be applied or prescribed, a complete vascular screening is needed to avoid skin complications from arterial insufficiency. This is especially important for patients who have a history of smoking, diabetes, or age over 60, all of whom are at higher risk for arterial insufficiency. See Chapter 4, Vascular Wounds, for information on vascular screening and guidelines for lower extremity compression.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

CLINICAL CONSIDERATION

In addition to the specific strategies for CVI and lymphedema, the plan of care should address all of the impairments that negatively impact joint ROM and muscle strength. In both venous and lymphatic dysfunction, muscle contraction plays a critical role in assisting fluid transportation.

The purpose of all of the interventions is to increase reabsorption at the venous/lymphatic capillaries, decrease the rate of capillary filtration, facilitate movement of the fluid from the distal superficial veins to the deep veins via the perforators, and assist lymphangion contraction. The interventions work together to increase the negative pressure gradient within the lymphatic and vascular vessels, thus promoting fluid movement from distal to proximal. It is the gradient, or difference in distal and proximal pressures, that forces the fluid flow in a specific direction. In the venous system, the venous pump creates 0 mmHg pressure in the deep veins so that fluid in the superficial veins moves to the deep veins, then proximally to the central system via the vena cava (see **TABLE 5-8**).

TABLE 5-8

Common Edema Diagnoses and Recommended Interventions

	AROM/Joint Mobs	Compression Garments or Bandages	Muscle Strengthening	Gait Training	MLD/Mobilization	Breathing Exercises	Aerobic Exercises	Skin Care	Patient Education
CVI	x	x	x	x				x	x
Secondary lymphedema	x	x	x	x	x	x	x	x	x
Lipedema		x	x	x	x	x		x	x
Orthopedic conditions with edema	x	x	x	x*	x	x		x	x
CVI/lymphedema	x	x	x	x	x	x	x	x	x

*Gait training is conditional, depending on the injury and weight-bearing status.

The selection of intervention(s) is individualized based upon the evaluation of the patients' activities and participation restrictions and not just the edema diagnosis.

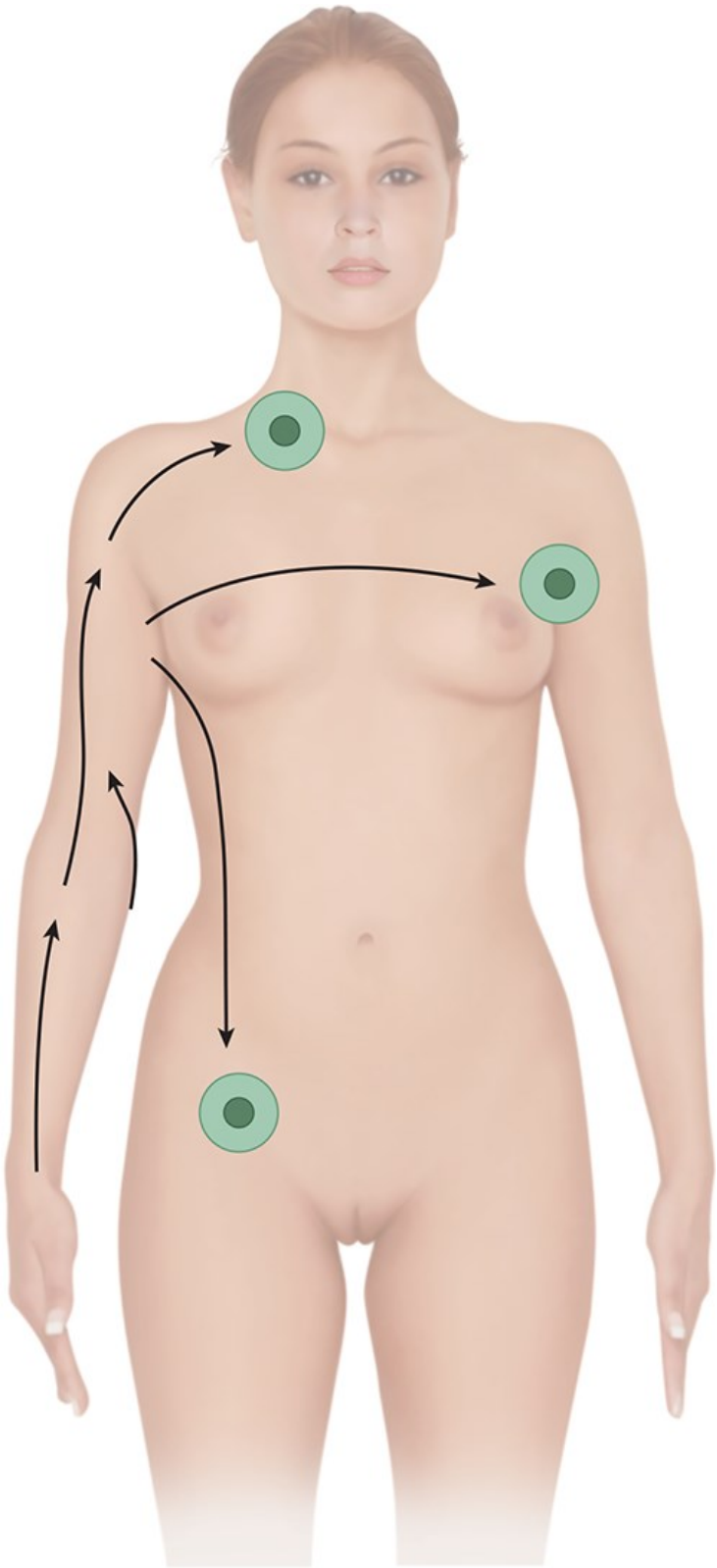
Lymph transport is dependent upon a negative pressure gradient within all of the lymphatic vessels. In the lymphatic system, the negative pressure in the thoracic duct and right lymphatic duct allows the *fluid* to move from distal to proximal (**FIGURE 5-30**). In order to accomplish this, treatment has to begin centrally and proximally and progress distally as each lymphatic component reaches a state of negative pressure. The negative pressure in the lymphatic capillaries facilitates movement of fluid from the interstitial tissue into the lymphatic system (deep to superficial). Lymph transport is dependent upon a negative pressure gradient within all of the lymphatic vessels. Therefore, treatment is predicated upon creating a negative pressure gradient in the proximal vessels (ie, the thoracic duct and right lymphatic duct) first, and then creating a negative pressure gradient in the sequential vessels, proximal to distal.

FIGURE 5-30

Flow of lymphatic fluid during manual lymphatic mobilization Lymphatic fluid flows from deep to superficial, distal to proximal as a result of negative pressure created by manual lymphatic mobilization. The circles represent lymph node regions: supraclavicular, axillary, and inguinal. The arrows demonstrate potential lymph drainage pathways for an individual with right upper extremity lymphedema secondary to breast cancer. The

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

therapist must decide if there are any contraindications to mobilizing lymph fluid to these alternate lymph node regions.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Manual Lymphatic Drainage Mobilization

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Manual lymphatic mobilization (MLD mob) is a specialized massage technique that stretches the superficial lymph vessels in a specific direction, thereby stimulating lymphangion contraction. The technique requires just enough pressure to stretch the skin (termed the "on phase") and then a smooth release of the skin (termed the "off phase") without the clinician pushing the skin back to its original starting point. The direction of the skin stretch governs the direction of lymph flow. The strokes are smooth and rhythmical in nature (**FIGURE 5-31**). Seven to ten repetitions of each stroke are performed in each location, beginning with stimulation of the central vessels, and then progressing sequentially, proximal to distal, along the extremity while maintaining the skin stretch toward the desired endpoint of fluid flow. MLD is followed by compression to help prevent return of fluid to the previous state of congestion.[34,35]

CLINICAL CONSIDERATION

When performing manual lymphatic mobilization on a patient who has congestive heart failure, the clinician must be cautious not to overload the heart with fluid that is being moved from the periphery to the thoracic duct. Evidence of overload can be assessed using the Borg Scale of Perceived Exertion, monitoring urinary output, and observing for signs of acute congestive heart failure. Caution may involve using limited interventions (eg, only MLD or only compressions, but not both) until the patient response to treatment is evaluated.[38]

FIGURE 5-31

Manual lymphatic drainage mobilization MLD mob requires specialized training and practice to be effective in moving lymphatic fluid from distal to proximal by first creating negative pressure in the proximal lymphatic structures, as well as moving the fluid from the deep tissue to the superficial lymphatic capillaries. In order for MLD to be effective, a negative pressure gradient is generated with diaphragmatic breathing and stimulation of the abdominal, supraclavicular, and cervical lymph node chains. The trunk is treated next, followed by the affected extremity proximal to distal. At the end of the treatment, the affected limb can be treated distal to proximal. MLD should be repeated with stimulation of cervical and supraclavicular lymph nodes and diaphragmatic breathing exercises. As with any intervention, the therapist must determine if any contraindications are present.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Compression Therapy

In addition to mobilizing the fluid, compression is used to restore Starling's Law of Equilibrium to normal, and thereby reduce the visible edema. This is accomplished by reducing the capillary filtration rate, increasing the reabsorption rate, and providing support to lymph transportation. **TABLE 5-9** lists the different types of bandage systems available and the indications for each type of compression.[35–37]

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-9

Types of Bandage Systems and Indications

Long stretch bandages: provide high pressure at rest and low pressure with activity. Goal is to move fluid into the deep venous system. *Indications*: component of multilayer compression for CVI *Contraindications*: ABI <0.8, vasculitis, lymphedema
Short stretch bandages: provide high pressure with activity and low pressure at rest. Goal is to decrease filtration and increase reabsorption and transportation of lymphatic fluid. *Indications:* lymphedema, lipedema, CVI with arterial compromise (ABI 0.5–0.8), traumatic edema, post-surgical swelling *Contraindications/precautions:* ABI <0.5, insensate extremity, uncompensated congested heart failure, cellulitis (unless being treated)

For lymphedema management, bandages are worn 20 out of 24 hours, thus allowing time out of the bandages each day for skin care, skin inspection, and laundering of the bandages (see **FIGURES 5-32** to **5-34**). The most effective plan of care involves wearing the bandages daily until the reduction has stabilized, at which point the patient is transitioned to a compression garment. Criteria for stabilized edema reduction include remodeling of the subcutaneous fibrosis, pliable tissue with no induration, and no further decrease in girth for 7–10 days.

FIGURE 5-32

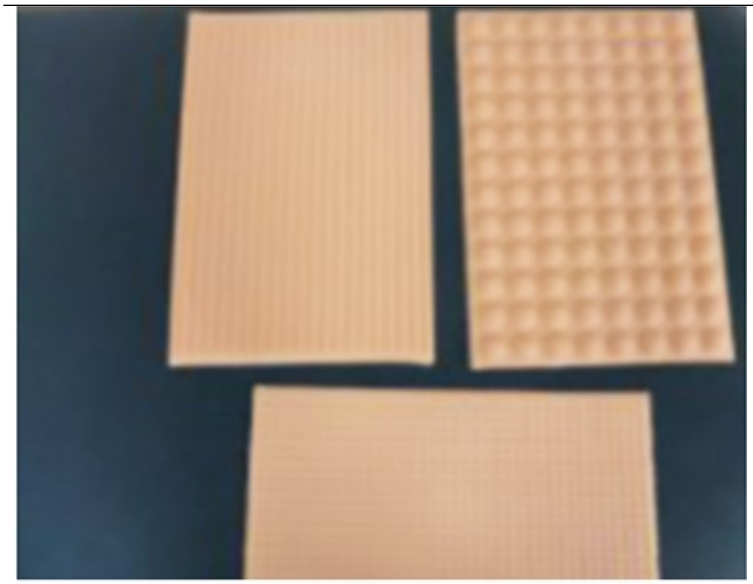
Components of a compression bandage kit A compression bandage kit includes foam, narrow gauze rolls for the fingers or toes, soft cotton padding for the first layer against the skin, and short stretch bandages of various widths. Narrow bandages are used distally, and progressively wider bandages used as the application progresses proximally.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-33

Foam used with compression Convoluted foam is used under compression bandages to help soften the hard fibrotic subcutaneous tissue that can develop with prolonged chronic edema.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-34

Compression bandages on an upper extremity Upper extremity compression begins with wrapping the fingers with narrow gauze in multiple layers, then the hand in narrow short stretch bandages, and then the forearm and arm, using a figure-8 or chevron pattern with wider bandages.

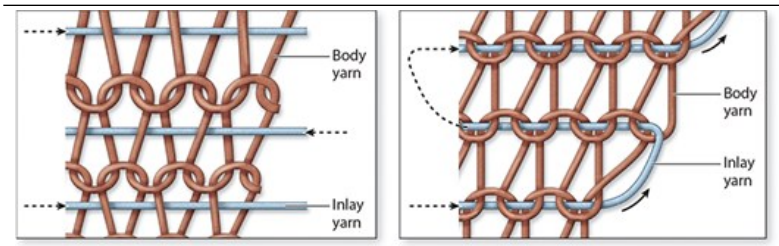
Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Selection and measurement for optimal garments are individualized to facilitate compliance with daily wear. Garments may be custom fit or over the counter; however, proper fit is of utmost importance. Generally, the larger and more fibrotic extremity requires the heavier garment with more coarse fabric. **FIGURES 5-35** and **5-36** illustrate the flat-knit and circular-knit construction used for compression garments. Length of the garment is dependent upon the extent of the edema (eg, if the edema is limited to below the knee, then a knee-high garment is appropriate; if the edema extends into or above the knee, a thigh-high or pantyhose garment is advised). Garments have a limited shelf-life of effective compression, ranging from 3 to 6 months, and need to be reordered according to manufacturer's guidelines (**FIGURES 5-37** and **5-38**). Optimally, daily washing of the garment is recommended.

FIGURE 5-35

Flat-knit construction of compression garments Flat-knit fabric is composed of the body yarn and the inlay yarn, and the tightness of the knit determines the coarseness of the fabric. Garments made of flat-knit fabric need to be stitched and are usually custom fit for better shape conformity. The total number of needles can be increased or decreased to produce variation in width and shape. The garments are generally coarser, less cosmetic, and stiffer; however, they are also better at bridging skin folds and less likely to cut the skin or cause a tourniquet effect.

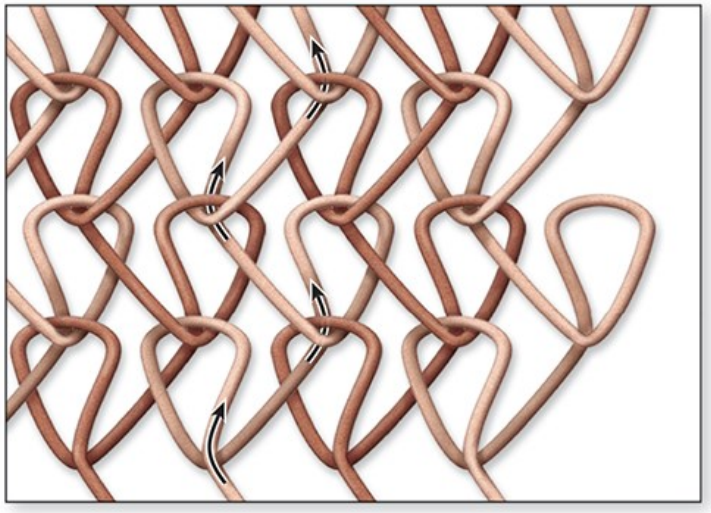
Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-36

Circular knit of compression garments Circular knit uses a fixed number of needles to produce a thinner fabric that is more cosmetically acceptable. It reduces the range of distortion but also increases the risk of creating a tourniquet effect if not properly fitted or donned.



Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-37

Toe garment The toe cap is an example of a garment made with flat knit fabric.

©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

FIGURE 5-38

Upper extremity compression garment The upper extremity garment is an example of construction with circular knit fabric.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Pumps

Compression pumps may be used as a complement to treatment of both venous and lymphatic pathologies (**FIGURE 5-39**). Venous pumps were designed based on the physiology of the venous system (intended to increase the venous return by generating high pressure in mmHg to promote reabsorption into the venous capillaries and facilitate flow through the superficial and deep veins), whereas the lymphatic pumps were designed based on the physiology of the lymphatic system (intended to increase the lymphatic circulation, central first and progressing sequentially proximal to distal). The lymphatic pump creates a negative pressure gradient within the thoracic duct, which then allows lymph fluid to move from an area of high pressure to low pressure. In other words, the proximal anatomical structures of the lymphatic system have to be emptied before the distal structures will drain.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Studies have not demonstrated return of protein molecules with either of the pump systems[39]; however, research has demonstrated that the lymphatic pumps are more effective in reducing lymphedema than the venous pumps.[34,35,37]

FIGURE 5-39

Intermittent compression pump for chronic venous insufficiency The intermittent compression pump is an adjunct therapy for peripheral edema. It is beneficial initially for decreasing edema before applying compression, and after resolution, for long-term edema management for those patients who cannot don/doff any type of garment.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

Exercise

Muscle contraction assists in lymphatic flow. The exercise sequence is based upon the physiological flow of lymphatic fluid; therefore, a lymph drainage exercise program begins with central exercises to stimulate the thoracic duct, cisterna chyli, and right lymphatic duct. Diaphragmatic breathing and/or aerobic exercises are the best exercises to achieve a negative pressure gradient in these central vessels. Exercises are individualized according to each patient's edema presentation and the direction of flow that is required for optimal outcomes. For example, a left UE breast cancer patient would start with exercise to move the fluid from the left upper quadrant to the right axillary lymph nodes and down to the left inguinal lymph nodes. Examples of exercise progression for the upper and lower extremity are listed in **TABLE 5-10**.[40]

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

TABLE 5-10

Exercise Programs for the Upper and Lower Extremities

Upper extremity: Diaphragmatic breathing Abdominal exercises Neck rotations Shoulder circles Shoulder to hand Pectoralis major ex (PNF diagonal D1, horizontal humeral adduction) Scapular adduction (rowing, scapular retraction) Latissimus (modified pull-down) Internal/external rotation Forward flexion to 90° Elbow flexion/extension Wrist flexion/extension/circles Finger flexion/extension/abduction/adduction Begin the exercises for the upper extremity by working from the shoulder to the hand, and then work distal to proximal with emphasis on shoulder rotation, circles, and breathing.	**Lower extremity:** Diaphragmatic breathing Abdominal exercises Neck rotations Shoulder circles Gluteal exercises (bridging) Inguinal lymph node stimulation (knee to chest) Anterior/posterior/lateral/medial thigh extension (open chain SLR in all 4 planes) Knee flexion/extension Ankle (ankle pumps, circumduction) Progress to closed chain exercises: Marching in place Modified squats Heel/toe raises Modified lunges Side-stepping Grapevine side to side Walking backwards End with inguinal node stimulation and diaphragmatic breathing.

Frequency, intensity, repetitions, and rest periods are based on patient level of physical fitness and exercise tolerance in order to prevent overloading the lymphatic system. All patients are advised to participate in a cardiovascular exercise program to stimulate the thoracic duct and cisterna chyli and to improve overall conditioning. Increased numbers of repetitions and weights, as well as shorter rest periods, may be added as the patient improves and the lymphedema decreases.

CLINICAL CONSIDERATION

Aquatherapy is contraindicated for any patient with an open wound, fragile skin, or insensate feet. Water exercises are beneficial for patients with very heavy limbs; however, meticulous skin care is required because of the effects of the chlorine. Aqua shoes are recommended for patients with lower extremity lymphedema in order to reduce the risk of infection; however, any bony deformities of the feet and the interdigital spaces need to be inspected after every exercise session.

There are no special diets or restrictions for upper and lower extremity lymphedema; however, maintaining a normal BMI is strongly encouraged.

Skin Care

Consequences of poor skin hygiene can be cellulitis or erysipelas (defined as an infection of the upper dermis and superficial lymphatics). With lymphedema the skin becomes dry, flaky, and cracking, which may provide a portal for bacteria to enter the skin. The first line of defense is the hydrolipid layer in the epidermis. When the tissue is congested with lymph fluid, the white blood cells may not reach the pathogens to phagocytose the bacteria. In addition, the decrease in transport capacity does not allow for adequate removal of bacterial debris that is present. If the skin is weeping, the moisture

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

accentuates the risk for bacterial colonization and possible infection. Following are recommendations for lymphedema skin care[41]:

- Moisturize with low pH, fragrant-free, hypoallergenic lotion, eg, a ceramide-containing emollient.
- Inspect the skin, interdigital spaces, and skin folds for cracks, blisters, and skin tears.
- Wash the skin daily with a mild hypoallergenic soap or moisturizing soap substitute; dry thoroughly with a clean towel.
- Seek podiatric care if the patient cannot reach the toenails.
- Do not cut cuticles (may cause skin openings that become infected); keep moisturized and pushed back.
- Wear protective gloves and shoes during activities that may cause minor trauma (eg, gardening, cooking); use an electric razor for shaving.
- Avoid overexposure to the sun because of the tendency to sun burn and blister.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

CASE STUDY

CONCLUSION

DISCUSSION QUESTIONS

1. What are your preferred interventions and the parameters for each of the interventions?
2. What are your outcome measures?

The patient had CVI with secondary lymphedema. There was concern for arterial insufficiency due to his comorbidities; however, the ABI was sufficient for short stretch compression bandages (**FIGURE 5-40**). Compression was indicated only to the knee because the diagnosis was CVI with secondary lymphedema (note that the edema stops at the knee). The patient was on Lasix, which was a concern for fluid overload of the kidneys. After consultation with the MD regarding his medication, Lasix was increased and the patient and his family were trained to monitor body weight, orthostatic hypotension, and increased need to void.

Treatment goals:

Decrease edema by 50–75%.

Increase hip and ankle ROM.

Increase gluteal muscles to alleviate the trendelenburg gait.

Functional goals:

Patient will be able to ambulate with heel/toe sequence sufficient to activate venous pump.

Patients will be able to play golf.

Interventions:

MLD

Short stretch bandages to knee

Exercise:

Supine ROM exercises proximal to distal with progression to weight bearing as strength and balance improved.

Patient was seen for 32 visits over a 4-month period.

Outcomes:

Improved 6-minute walk to 500 yards.

Performed 7 sit-to-stand in 30 seconds with UE assist.

Ambulated with rolling walker using heel-toe gait.

Managed edema with knee-high garments.

Practiced putting with stand by assist of one person for safety.

FIGURE 5-40

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

Case study after treatment After treatment with MLD and compression therapy, the patient was transitioned to compression stockings. Note the indentation in the soft tissue on the right lower leg just below the knee. Such indentations are a result of inappropriate donning of the garment, resulting in restriction of lymphatic and venous return in places where there is high compression. This in turn causes a tourniquet effect with resulting increased edema below the constriction. This is observed in the larger size of the right calf as compared to the left.

Source: Rose L. Hamm: *Text and Atlas of Wound Diagnosis and Treatment*, 2e
Copyright © McGraw-Hill Education. All rights reserved.

SUMMARY

Peripheral edema is a symptom of an underlying pathology that must be identified in order to develop an effective plan of care for the patient. Differential diagnosis requires information on all of the body systems and information from all of the medical specialists involved in the patient's care. The goal is to determine if the edema is caused by lymphatic dysfunction, venous insufficiency, organ failure, or medications. An effective care plan requires treatment of the underlying disorders and sometimes changes in medication. Although there are overlaps in the treatment of lymphedema and CVI (eg, exercise and compression therapy), there are specific differences; thus, management of the specific system (lymphatic versus venous) is essential in order to have the optimal outcomes.

REFERENCES

1. Foldi M, StroBenreuther R. Lymph formation and lymph flow: physiological lymph drainage. In: Foldi M, StroBenreuther R. eds. *Foundations of Manual Lymph Drainage*. 3rd ed. St. Louis, MO: Elsevier Mosby; 2005:28–38.

2. Marieb EN. *Human Anatomy & Physiology*. 6th ed. London: Pearson; 2004:736–738, 737, 773.

3. Rockson SG. Current concepts and future directions in the diagnosis and management of lymphatic vascular disease. *Vasc Med*. 2010;15(3):223–231.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

doi:10.1177/1358863x10364553.

4. Little RC, Ginsburg JM. The physiologic basis for clinical edema. *Arch Intern Med*. 1984;144:1661–1664. [PubMed: 6466021]

5. Tiwari A, Cheng K, Button M, Myint F, Hamilton G. Differential diagnosis, investigation, and current treatment of lower limb Lymphedema. *Arch Surg*. 2003;138(2):152–161. [PubMed: 12578410]

6. Sontheimer DL. Peripheral vascular disease: diagnosis and treatment. *Am Fam Physician*. 2006;73(11):1971–19767. [PubMed: 16770929]

7. Zuther JE, Norton S. *Lymphedema Management: The Comprehensive Guide for Practitioners*. 4th ed. New York, NY: Thieme Medical Publishers; 2018.

8. Foldi E, Foldi E. Physiology and pathophysiology of the lymphatic system. In: Foldi E, Foldi M, eds. *Foldi Textbook of Lymphology*. 2nd ed. Munich: Elsevier; 2006:179–222.

9. Cormier JN, Askew RL, Mungovan KS, Xing Y, Ross MI, Armer JM. Lymphedema beyond breast cancer: a systematic review and meta-analysis of cancer-related secondary lymphedema. *Cancer*. 2010;116(22):5138–5149. [PubMed: 20665892]

10. Norman SA, Localio AR, Potashnick SL, et al. Lymphedema in breast cancer survivors: incidence, degree, time course, treatment and symptoms. *J Clin Oncol*. 2009;27(3):390–397. [PubMed: 19064976]

11. Lucci A, McCall LM, Beitsch PD, et al. Surgical complications associated with sentinel lymph node dissection (SLND) plus axillary lymph node dissection compared with SLND alone in the American college of Surgeons Oncology Group Trial Z0011. *J Clin Oncol*. 2007;25:3657–3663. [PubMed: 17485711]

12. Shin BW, Sim YJ, Jeong HJ, Kim GC. Lipedema, a rare disease. *Ann Rehabil Med*. 2011;35:922–927. [PubMed: 22506222]

13. Child AH, Gordon KD, Sharpe P, et al. Lipedema: an inherited condition. *Am J Med Genet A*. 2010;152A:970–976. [PubMed: 20358611]

14. Warren AG, Janz BA, Borud LJ, Stavin SA. Evaluation and management of fat leg syndrome. *Plastic Recontruct Surg*. 2007;119:9e–15e.

15. International Society of Lymphology. The diagnosis and treatment of peripheral lymphedema: consensus document of the International Society of Lymphology. *Lymphology*. 2003(36):84–91.

16. Newman B, Lose F, Kedda MA, et al. Possible genetic predispostion to lymphedema after breast cancer. *Lymph Res Bio*. 2012;10(1):2–13.

17. Armer JM, Stewart BR. A comparison of four diagnostic criteria for lymphedema in a post-breast cancer population. *Lymphedema Res Biol*. 2005;3:208–217.

18. Gartner R, Jensen MB, Kronborg L, et al. Self-reported arm lymphedema and functional impairment after breast cancer treatment: a nationwide study of prevalence and associated factors. *Breast*. 2010;19:506–515. [PubMed: 20561790]

19. Mortimer PS. Managing lymphedema. *Clin Dermatol*. 1995;13:499–505. [PubMed: 8665461]

20. Ely JW, Osheroff JA, Chambliss ML, Ebell MH. Approach to leg edema of unclear etiology. *J Am Board Fam Med*. 2006;19:148–160. [PubMed: 16513903]

21. Levenhagen K, Davies C, Perdomo M, Ryans K, Gilchrist L. Diagnosis of upper quadrant lymphedema secondary to cancer: clinical practice guidelines from the Oncology Section of the American Physical Therapy Association. *Phys Ther*. 2017;97(7):729–745. Available at: https://doi-org.ezproxy.cihe.edu.hk/10.1093/ptj/pzx050. Accessed July 23, 2018.

22. Megens AM, Harris SR, Kim-Sing C, McKenzie DC. Measurement of upper extremity volume in women after axillary dissection for breast cancer. *Arch Phys Med Rehabil*. 2001;82:1639–1644. [PubMed: 11733875]

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

23. Szuba A, Shin WS, Strauss HW, Rockson S. The third circulation: radionuclide lymphoscintigraphy in the evaluation of lymphedema. *J Nucl Med*. 2003;44:43–57. [PubMed: 12515876]

24. Partsch H. Assessment of abnormal lymph drainage for the diagnosis of lymphedema by isotopic lymphangiography and by indirect lymphography. *Clin Dermatol*. 1995;13(5):445–450. [PubMed: 8665455]

25. Rasmussen JC, Tan I, Marshall MV, Fife CE, Sevick-Muraca EM. Lymphatic imaging in humans with near-infrared fluorescence. *Curr Opin Biotechnol*. 2009;20(1):74–82. [PubMed: 19233639]

26. Marshall MV, Rasmussen JC, Tan I, et al. Near-infrared fluorescence imagin in humans with indocyanin green: a review and update. *Open Surg Oncol J*. 2010;2(2):12–25. [PubMed: 22924087]

27. Pusic AL, Cemal Y, Albornoz C, et al. Quality of life among breast cancer patients with lymphedema: a systematic review of patient-reported outcome instruments and outcomes. *J Cancer Surviv*. 2013;7(1):83–92. [PubMed: 23212603]

28. Devoogdt N, DeGroef A, Hendrickx A, et al. Lymphoedema functioning, Disability and Health Questionnaire for Lower Limb Lymphoedema (Lymph-ICF-LL): reliability and validity. *Phys Ther*. 2014;94(5):705–721. [PubMed: 24415775]

29. Blome C, Augustin M, Heyer K, et al. Evaluation of patient-relevant outcomes of lymphedema and lipedema treatment: development and validation of a new benefit tool. *Eur J Vasc Endovasc Surg*. 2014;47(1):100–107. [PubMed: 24239143]

30. Augustin M, Condo Montero E, Hagenstrom K, Herberger K, Blome C. Validation of a short form FLQA-LS quality of life instrument for lymphedema. *Brit J Dermatol*. 2018;21. Available at: http://www.ncbi.nlm.nih.gov/pubmed/29927481. Accessed July 23, 2018.

31. Bunke N, Brown K, Bergan J. Phlebolymphemeda: usually unrecognized, often poorly treated. *Perspect Vasc Surg Endovasc Ther*. 2009;21(2):65–68. [PubMed: 19767404]

32. Mayrovitz HN. The standard of care for lymphedema: current concepts and physiological considerations. *Lymphat Res Biol*. 2009;7(2):101–108. [PubMed: 19522678]

33. Oremus M, Walker K, Dayes I, Raina P. *Diagnosis and Treatment of Secondary Lymphedema*. AHRQ Technology Assessment Report Project ID: LYMTO9O8; 2010.

34. Huang T-W, Tseng S-H, Lin C-C, et al. Effects of manual lymphatic drainage on breast cancer-related lymphedema: a systematic review and meta-analysis of randomized controlled trials. *World J Surg Oncol*. 2013;11:15. Published online January 24, 2013. doi:10.1186/1477-7819-11-15. Accessed June 21, 2018.

35. Kerchner K, Fleischer A, Yosipovitch G. Lower extremity lymphedema update: pathophysiology, diagnosis, and treatment guidelines. *J Am Acad Dermatol*. 2008;59:324–331. [PubMed: 18513827]

36. Preston N, Seers K, Mortimer P. Physical therapies for reducing and controlling lymphedema of the limbs. *Cochrane Database Syst Rev*. 2008(4):CD003141.

37. Rabe E, Partsch H, Hafner J, et al. Indications for medical compression stockings in venous and lymphatic disorders: an evidence-based consensus statement. *Phlebology*. 2018;33(3):163–184. [PubMed: 28549402]

38. Vaassen MM. Manual lymph drainage in a patient with congestive heart failure: a case study. *Ostomy Wound Management*. 2015;61(10):38–45.

39. Feldman JL, Stout NL, Wanchai A, Stewart BR, Cormier JN, Armer JM. Intermittent pneumatic compressin therapy: a systematic review. *Lymphology*. 2012;45:13–25. [PubMed: 22768469]

40. Chang CJ, Cormier JN. Lymphedema interventions: exercise, surgery and compression devices. *Seminars Oncol Nursing*. 2013;29(1):28–40.

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility

41. Fife C, Farrow W, Hevert AA, et al. Skin and wound care in lymphedema patients: a taxonomy, primer, and literature review. *Adv Skin Wound Care*. 2017;30(7):305–318. [PubMed: 28617750]

42. Perdomo M, Davies C, Levenhagen K, Ryans K. Breast Cancer Edge Task Force Outcomes: Assessment Measures of Secondary Lymphedema in Breast Cancer Survivors. *Rehab Oncol*. 2014;32(1):22–35. Photographs used with permission.

43. From Megens, AM, Harris SR, Kim-Sing C, McKenzie DC. Measurement of Upper Extremity Volume in Women After Axillary Dissection for Breast Cancer. *Arch Phys Med Rehabil*. 2001;82:1639–1644. [PubMed: 11733875]

Downloaded 2022-3-23 7:53 A Your IP is 165.84.189.140
Chapter 5: Lymphedema, Marisa Perdomo; Rose L. Hamm
©2022 McGraw Hill. All Rights Reserved. Terms of Use • Privacy Policy • Notice • Accessibility